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THE BRITISH JOURNAL
OF
OPHTHALMOLOGY
JULY, 1922

COMMUNICATIONS

TABES AND OPTIC ATROPHY

BY

LESLIE PATON

LONDON

ON looking over a series of visual fields in tabetics, one cannot help being struck by the remarkable variability that they show and the marked disproportion that exists in different cases between the loss of visual acuity and the loss of visual field. Is there anything in the underlying pathological changes which will explain this variability? To determine this, it is necessary to see what has been learned of the pathology of tabes in general and more particularly of tabetic optic atrophy. I have tried to show in the following paper in how far the process as it concerns the optic nerve, resembles or differs from the process in other parts of the central nervous system. Of necessity, as the literature on the subject is immense, I can only refer to the more outstanding papers that have come to my notice, but I think these show how varied and discordant are the views held as to the nature of tabes.

It is obvious that if tabes is a clinical entity, a homogeneous disease, and not simply a heterogeneous collocation of manifestations of later syphilis, there must be some definite relationship in the pathology of the conditions that are found in different parts of the central nervous system; and the variations that occur must be due more to variations in the nature of the tissue attacked than to variations in the virus.

The Lesion in Tabes

In tabes, we have one of the most common diseases of the central nervous system, and yet one in which the pathological process at work is the subject of the most diverse views. Much of the difficulty is due to two causes; in the first place almost invariably the investigations have been made in cases where the disease has reached a most advanced stage, and secondly, as tabes is a late manifestation of syphilis, a patient with this lesion is likely to present many other lesions resulting from syphilitic infection. In consequence of these two facts, it becomes a very difficult matter to separate out and define which of the pathological changes found are essentially parts of the clinical entity tabes, and which are to be regarded as complications due to other syphilitic conditions. And this is more especially difficult since syphilis can produce so many diverse effects on the central nervous system, *e.g.*, meningitis, myelitis, and all the variety of lesions resulting from syphilitic vascular lesions. We find accordingly that when a writer is supporting any particular hypothesis, he always adduces a co-ordinate syphilitic complication to explain away all the facts that are not in accordance with the hypothesis he favours.

A. Position of Originating Lesion

The present-day theories of tabes may be subdivided into three main groups according to the position in which the pathological changes are supposed to begin.

1. The posterior columns.
2. The posterior root ganglion.
3. The posterior roots.

The placing of the origin of tabes in the posterior columns is a perpetuation of the older hypothesis of the disease. Strümpell's[47] support of it is based on three points:—

(*a*) Certain tracts in the posterior columns remain unaffected.

(*b*) The affection always shows a certain distribution involving more intense implication of certain zones and relative freedom of other regions which are equally of post root-fibre origin.

(*c*) The symmetrical nature of the distribution.

Flechsig[8] has supported the theory of the elective action of the tabetic virus on certain fibre tracts in the posterior columns on biological grounds. He considers that there is variation in the vulnerability of tracts according to the variation in the time of development of their myelin sheaths (*v.* also Kaufmann[12]).

Both these writers support the hypothesis of a systemic elective action of a tabetic toxin (meta-syphilitic) directly on nerve fibres of the posterior column, *i.e.*, a parenchymatous action of the virus.

In the second hypothesis, the posterior root ganglion is sug-

gested as the point of origin of the degeneration, and tabes is regarded as a typical neuronic degeneration commencing in the ganglion cells of the posterior root ganglion. The degeneration of the posterior roots and posterior column tracts is, on this theory, regarded as secondary to the degeneration of the ganglion cells. This hypothesis has been strongly supported by Stroebe[44], Marinesco[20] and Köster[14], and again the toxin is supposed to act on parenchymatous tissue.

It is on the third hypothesis of a primary affection of posterior root fibres that most of the discussion in recent years has taken place.

According to the hypothesis of Obersteiner and Redlich[30], the posterior root fibres are primarily attacked as they pass through the pia mater to enter the posterior columns. There is at that point a diminution in the myelin sheath which renders the fibres more vulnerable. The bundles are, at this point, constricted partly by a meningitis and partly by arterio-sclerotic changes in the branch of the artery which comes into relationship with these entering bundles.

Orr and Rows[33] also are of opinion that the process has its starting place at the point of entry of the posterior root into the cord, but they do not agree with the Obersteiner-Redlich idea of a primary meningeal cause. They consider that it is primarily a parenchymatous degeneration, and that the reason that the toxin produces its effect at this particular place is that here the nerve fibres lose their neurilemmal sheath which blends with the pia, and at the same time the myelin sheath much diminishes in thickness, so that, generally, the nerve fibre is less protected than elsewhere. In addition, the main lymph channels enter the cord along with the posterior roots, and as they convey the virus the posterior roots are more exposed to its attack at this point. Their work on other infective conditions in the central nervous system has shown the importance of the lymph channels in the conveyance of infections, and especially the lymph channels which accompany the afferent nerves from the periphery to the cord.

According to Nageotte's[24] hypothesis, the lesion of the posterior roots is completely extra-pial, and the root is primarily attacked in the portion nearest the ganglion where the dura and arachnoid sheaths have joined up into one, but where there still exists a definite subarachnoid space, forming a pocket or recess which is carried further out along the posterior root than along the anterior root. The portion of nerve lying in this space, the " radicular nerve of Nageotte," is supposed to be the part of the nerve first affected. (*v.* Fig. 1.)

This theory of Nageotte has been very strongly supported by the recent work of Richter[38].

Nature of Pathological Lesion

The discovery of spirochaetes in the brain of paretics by Noguchi[25] in 1913 rendered it increasingly probable that sooner or later these would also be found in cases of pure tabes, and their localization would help to give a clue to the point of origin of the disease. The difficulty again was likely to lie in the other syphilitic complications that so frequently accompany and complicate tabes. If Richter's[38] discovery of spirochaetes in a few cases of tabes in the granulation tissue in the sub-arachnoid recess which surrounds the radicular nerve is confirmed, it will go far to solve the problem of tabes. Before dealing with Richter's views, I will briefly state some of the earlier hypotheses as to the nature of the pathological lesion.

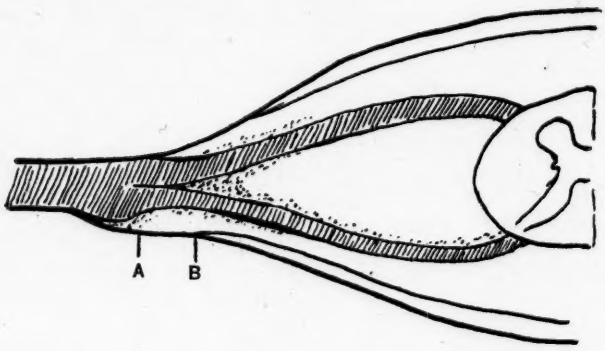


FIG. 1.
A—B RADICULAR NERVE (Nageotte).
To illustrate Nageotte's and Richters' Hypotheses.

I. That the condition is one of primary nerve fibre degeneration, brought about by the selective action of a meta-luetic toxin on certain nerve tracts, and that the proliferation of connective tissue and glial tissue and the other signs of chronic inflammatory change are secondary to and consequent on the nerve atrophy. This hypothesis can be sub-divided into two sub-groups :—

(*a*) Where the primary degenerative process takes place in the posterior columns.

(*b*) Where the primary degenerative process takes place in the posterior roots.

I have already referred to the work of Orr and Rows[33] which would place tabes under this heading. On the same lines runs the work of McIntosh, Fildes[21], Head and Fearnsides[11]. According to these writers, the essential features of syphilis are the same in

all its stages, and both interstitial and parenchymatous tissue may be affected. There may be evidence not only of perivascular proliferation and small celled infiltration, but also of intoxication of the parenchyma itself. " The difference between the interstitial tissues and the parenchyma is, that in the former the reaction is marked—proliferation and infiltration—while the degeneration is slight, and in the latter the reaction is trivial and the degeneration extensive."

This applies both to the acute and chronic stages of the disease. The parenchymatous tissue has little or no reparative power, and consequently degeneration in it means loss of function.

In the tertiary stages, the number of spirochaetes has markedly diminished, and " there is a remarkable lack of correlation between the amount of virus and the extent of the lesions. This disproportion is due to the poisoning of the tissues in the secondary stages producing a great increase in the susceptibility of the tissues to the virus (hyperallergie), so that the awakening into activity of even a small number of spirochaetes produces a very marked tissue reaction or tissue degeneration. These spirochaetes having escaped the general destruction which takes place towards the end of the secondary stages (which destruction may be caused by the development of antibodies or by drugs), may remain dormant for years until awakened into activity by some cause as yet obscure."

In dementia paralytica the pia is in a state of inflammation, showing infiltration by large numbers of lymphocytes and plasma cells and proliferation of the endothelium of the capillaries and small vessels. In addition to these changes in the mesoblastic tissues, there are changes in the ectoblastic tissues, degeneration of ganglion cells and increase of neuroglial cells. But the changes in the parenchyma are more marked than those in the interstitial tissue, *i.e.*, the disease is essentially a parenchymatous encephalitis.

In tabes, the essential lesion " also is due to an inflammatory reaction on the part of the nerve fibres and neuroglia induced by the presence of the spirochaeta pallida." " The degeneration of the nerve fibres following on this intoxication will occur in those parts only which have been deprived of the neurilemma." The writers adopt the theory of Orr and Rows as to the primary site of the lesion being where the neurilemmal sheath leaves the roots to blend with the pial sheath and the thinner nerve root passes into the cord tissues, and also with their theory that the lymph channels convey the virus to this position.

II. That there is a co-ordinate action of the virus on nerve fibres and supporting tissue, and that the changes in the two tissues are to that extent independent of one another.

Against this hypothesis, as Richter points out, there is the fact that there is never nerve degeneration without the presence of

granulation tissue, but there is frequently found well-marked development of granulation tissue preceding nerve atrophy, and it is only in the immediate neighbourhood of granulation tissue that nerve degeneration is found.

III. That the nerve degeneration is the consequence of the development of the granulation tissue, and is brought about partly by the disturbance of nutrition caused by granulation tissue developing in and blocking the normal lymph channels and finer connective tissue spaces by which the nerve fibres obtain their nourishment, and partly by the mechanical constriction of the fibres by the growth of neuroglial tissue, *i.e.*, the process is partly bio-chemical and partly mechanical.

According to Obersteiner and Redlich[30], this process of constriction and blockage takes place in the pial region, and is consequent on a primary meningitis, of which the evidence is the existence, not only of fibroblastic cells, but also of lymphocytes and plasma cells. To this exudative process is superadded a further constriction by arterio-sclerotic changes in a small branch of the posterior spinal artery.

In 1894 Nageotte[24] published his first paper, in which he described the changes as commencing round the nerve roots towards the outer portion where they come close together and lie in a common sheath. He describes the nerve roots as consisting of three parts :—

1. Inside the cord until they pierce the pia mater.
2. Lying more or less free and in several fascicles in the sub-arachnoid space. In this portion, they get a partial sheath of sub-arachnoid tissue which gradually thickens into a more definite lamella as the bundles fuse together to form the third portion.
3. Fused bundles joined up and surrounded with a sheath composed of combined dura and arachnoid which closes in on the combined anterior and posterior roots before the ganglion is reached. This last portion, which he calls *nerf radiculaire*, is, as I have already said, the point of origin of the lesion. Pathologically, both Nageotte and his latest supporter, Richter, describe the changes as commencing in the sub-arachnoid tissue surrounding this portion of the nerve.

In Richter's words[38]—" the recognisable nerve changes in tabes depend upon a direct lesion of the nerve root fibres in the area of Nageotte's radicular nerve caused by syphilitic granulation tissue which is produced in the lymph spaces of the connective tissue sheaths by the irritation of spirochaetes deposited there, and this spreading along the lymph channels in the nerve bundle causes local patches of disturbance."

The granulation tissue originally described by Nageotte in 1894 as an *infiltration embryonnaire*, results from the proliferation of

endothelial cells in the lymph spaces and of the fixed connective tissue cells. According to Richter, there are no cells of haematogenous origin, no lymphocytes nor plasma cells, but only pure fibroblastic cells. These first develop in the lymph spaces of the combined dural-arachnoid sheath, and from there the granulation tissue makes its way into the looser tissue of the sub-arachnoid space and invades the nerve bundles, making its way by lymph channels and spaces up the nerve towards the cord and down towards the ganglion.

The process in tabes differs from that found in general paralysis or tabo-paresis in the absence of haematogenous cells. In general paralysis and tabo-paresis, both lymphocytes and plasma cells are found invading the tissues, and in the perivascular lymph spaces.

Affections of Cranial Nerves other than the Optic Nerve

Of the cranial nerves other than the optic nerves the oculo-motor nerves are more frequently attacked in tabes than any other. The general opinion of the majority of workers has been in favour of regarding tabetic oculo-motor palsies as of nuclear origin. In the paper on " Ocular Palsies " I read in March, 1921, at the Royal Society of Medicine (*B.J.O.*, June, 1921), I stated this hypothesis somewhat baldly and dogmatically. The extent of the subject to be covered in that discussion must be my excuse. It is far from being generally accepted. If it be true, it places tabetic oculo-motor palsies in a different position from the tabetic affections of the cord. In none of the theories of tabes is the primary lesion supposed to be in the grey matter of the cord. The nearest analogous theory is that which places the primary lesion in the posterior ganglion cells. Again it is the nucleus of origin of an efferent nerve which is here supposed to be affected, and in the cord the afferent nerve is always the more affected and the anterior root shows a much greater resistance to the disease and is much later in yielding to it. On this point, the idea suggested by Sherrington[42], mainly based on the work of de Kleijn, that the fleeting diplopias of early tabes are due to disturbances in the afferent proprioceptive impulses from the oculo-motor muscles and muscles of the head and neck, should be remembered. The point is of importance as hinting that here also, as in the other tabetic lesions, the primary disturbance may be in afferent fibres. In the case of the eyes, however, the afferent impulses from the retina and from the semicircular canals predominate to such an extent over all other afferent impulses that the diplopia produced by disturbances of the latter is speedily compensated and proves but evanescent. This disturbance of afferent impulses probably plays no part in the production of the permanent oculo-motor palsies of late tabes. The

hypothesis that this is not primarily a nuclear degeneration but an affection of the nerve stem, has the support of many writers, *e.g.*, Oppenheim[32], Dejerine[4], Nonne[28] and Cassirer[2], and, more recently, Stargardt[43] and Richter[38].

The hypothesis of Stargardt is that the affection of the oculo-motor nerves commences by an infiltration of the sheath and vessels of the oculo-motor nerve. In the central nervous system, the Grenz-membrane shuts these off from the fibres themselves, but in the extra-cerebral nerve roots, the ecto-dermal Grenz-membrane is represented only by the sheath of Schwann surrounding individual fibres, so that the infiltrating tissue passes in among the nerve fibres. The primary point of invasion is where the nerve passes through the dura; and the infiltrating tissue contains cells of haematogenous origin, plasma cells and lymphocytes. In Richter's hypothesis, the process commences in the perineurium of the extra-cerebral portion of the nerve, and the granulation tissue developing in this area is of exactly the same nature as that which he finds in the posterior nerve roots. He admits that cells of haematogenous origin are to be found in the pial zone, but in the tissue actually invading the nerve, he finds only fibroblasts and no lymphocytes nor plasma cells. The difference between his findings and Stargardt's he ascribes to the fact that he was only dealing with pure tabetics, while Stargardt's cases included tabo-paretics and paretics.

Optic Atrophy

Various and discordant as are the theories advanced to explain the pathology of the changes in the posterior roots and columns, we find that the theories of optic atrophy are just as diverse. The possibilities may be briefly grouped into :—

1. A primary neuronic degeneration with secondary fibrosis :—

(*a*) Commencing in the ganglion cells (Gowers[10], Erb[7], Charcot[3], etc.).

(*b*) Commencing in the nerve fibres or axones (Uhthoff[45], etc.).

2. A primary peripheral and interstitial neuritis with secondary degeneration of nerve fibres.

(*a*) The primary changes commencing in the papilla (Elschnig[6]).

(*b*) The primary changes commencing in the pial and subpial regions in the peripheral portion of the nerve (Léri[16], Wilbrand and Saenger[49]).

(*c*) The primary changes both axial along the central vessels and peripheral in the subpial zone.

(*d*) The primary changes commencing in the optic foramen (a periostitis and pachymeningitis resulting in strangling of the nerve (Schlagenhaufer).

(*e*) The primary changes commencing in the intra-cranial portion of the nerve including the chiasma and optic foramen and mainly in the pial and subpial zone (Stargardt and Richter).

3. The interstitial changes and the parenchymatous changes in the nerve are co-ordinate results of the activity of the tabetic virus and not mutually dependent on one another.

Hypotheses of Primary Neuronic Degeneration

Parsons[34] in " The Pathology of the Eye," Vol. IV, p. 1344, gives what may be taken as the commonly accepted view up to the end of the nineteenth century—" the primary seat of the disease is in the retina, commencing in the ganglion cells and nerve fibre layer. In the nerve, the atrophy is generally most marked near the globe, diminishing towards the cerebrum; changes in the chiasma and tracts are absent in the early, and are slight even in the later stages." This view of a primary neuronic degeneration commencing in the ganglion cells was held by Erb, Charcot, Gowers, Popow[36], Moxter[23], v. Michel, de Grosz[46], Klippel[13], Gliksmann[9], Wagenmann[48] and Coppez, but the arguments against it are numerous. These were summarized by Léri in 1904. Holden in 1899 argued that the changes in the ganglion cells were secondary to degeneration of nerve fibres. Léri found numerous ganglion cells intact in the retina where every fibre in the nerve had disappeared, and in all cases there was a marked disproportion between the ganglion cell and nerve fibre layer degeneration and the degeneration in the nerve itself. From the examination of the retina and nerve in eleven cases of blind tabetics, he concluded that the origin of tabetic atrophy could not be in the retina itself.

Stargardt summarises his conclusions as regards the retinal origin of the optic atrophy in tabes and general paralysis in the following eight clauses:—

1. The retina never shows, during the course of optic atrophy in tabes or general paralysis, a special pathological histology. The changes which occur in the retina are throughout analogous with those which occur in any descending atrophy, such as may result from the pressure of an arterio-sclerotic internal carotid. The pyknomorphic ganglion cells are found in both conditions indicating a definite reparative attempt. Also the slight changes in the inner nuclear layer are similar in character.

(Nissl's pyknomorphic ganglion cells are supposed to indicate a reparative effort in the nervous tissues. The nucleus, which in degeneration has become pushed to the side, gets back to the centre of the cell. Immediately round it, large Nissl granules develop, and then later similar large Nissl granules develop in the periphery. The Nissl granules are all larger and more numerous than in the ordinary ganglion cell.)

2. The retinal changes in tabes and general paralysis are in all essentials similar to those found after experimental section of the nerve. In animals, however, there are no pyknomorphic ganglion cells found, nor changes in the inner nuclear layer.

3. There are no changes found in the outer retinal layers such as are found after certain poisons, *e.g.*, quinine, aspidium, filix mas, and atoxyl.

4. There are no changes found in the retina unless there are undoubted changes in the optic nerve.

5. In the earliest stages of optic atrophy, no changes are found in the retina.

6. In partial optic atrophies, the retinal changes found are strictly confined to the portions which correspond to the part of the nerve affected.

7. In partial optic atrophy, the amount of retinal change found is relatively less than the amount of optic nerve change.

8. After complete optic atrophy, quite normal ganglion cells, though few in number, can be found in the retina.

His general conclusion from retinal examinations is that the retina cannot be the seat of the primary change in tabetic atrophy.

The hypothesis of a neuronic degeneration beginning in the nerve fibres in the nerve itself has become widely accepted amongst ophthalmologists on account of its advocacy by Uhthoff in his article in Graefe-Saemisch, and later by Wilbrand and Saenger[49] in "Neurologie des Auges," Vol. V, p. 585. Uhthoff describes the process as commencing in a fatty degeneration and absorption of the myelin sheaths followed by changes in the axis cylinders. These first show varicosities and subsequently also degenerate, leaving only the fine threads of collapsed nerve fibre sheaths. In the later stages, all that is left is fatty drops, myelin drops, and amyloid bodies.

The changes in the interstitial tissue follow on these parenchymatous degenerations. The finer septa and processes of supporting tissue disappear and the larger septa become more swollen. There is no evidence of any chronic inflammatory process producing these changes, neither cell infiltration nor nuclear proliferation, only that with the disappearance of the nerve fibres the larger septa become more prominent. It is only in the latest stages that glial proliferation comes into evidence. Such, briefly, is Uhthoff's description of the process which he regards as a neuronic degeneration commencing in the nerve fibres and due to a tabetic or metasyphilitic toxin. He considers that the process commences in and is most marked in the peripheral portion of the nerves and diminishes in intensity towards the intra-cranial portion and the chiasma, and is mostly absent above the chiasma. "A primary starting point of the degeneration in the chiasma or in the optic

tract, or in the primary centres of the optic nerves, seems to be excluded." He considers that the early appearance of disc changes points to the peripheral origin of the lesion.

Hypotheses of Primary Peripheral and Interstitial Neuritis

I now come to the consideration of the hypotheses which regard the primary changes as taking place in the peripheral and interstitial tissues. Of the sub-divisions of this group, three require only very brief mention.

Elschnig[6], in 1899, after the examination of a case of tabes with marked atheroma, suggested the hypothesis that " the cause of the optic atrophy could be found in the overgrowth of thick glial tissue in the disc replacing the nerve fibres." (" Das dichte Gliagewebe, welches das Nervenfasergewebe in der Papille substituiert, wohl ganz in diesem Sinne als Ursache des Schwundes der Nervenfasern angesehen werden könnte.")

Elschnig, in advancing this hypothesis, seems to have been influenced greatly by Obersteiner and Redlich's hypothesis of the origin of tabetic degeneration by strangulation of the posterior root in its passage through the pia.

Schlagenhaufer[41] ascribed the optic atrophy to a strangling of the nerve in its passage through the optic foramen in consequence of a syphilitic periostitis or pachymeningitis. Probably a process somewhat similar to this is the cause of the optic atrophy in oxycephaly, but in that case, there is evidence also in some cases of a true optic neuritis. The interest of this hypothesis also lies in its analogy with that held by Obersteiner and Redlich for tabes.

There seem to be very few authorities prepared to support an axial origin of the interstitial changes. Peltesohn[35] found in one patient with commencing tabes, a patch of central atrophy in the optic nerve. Clinically, the great rarity of cases of central scotoma is a strong argument against it, and a still stronger argument against it is the fact that the retinal vessels show little or no alteration in their calibre, even when the atrophy is very advanced and the sight almost gone.

The great majority of writers in favour of the interstitial theories describe the process as commencing in the periphery of the nerve. Léri[16], in his paper published in 1904, recorded the results of his examination of the optic nerves of 84 cases. Of these, 21 were tabetics, 19 were completely blind and 2 not completely blind. Three were cases of blind general paralytics, 16 tabetics and 18 general paralytics without evidence of visual loss, were examined. The rest were mostly cases of different forms of syphilis affecting the central nervous system. He describes in detail the macroscopic and microscopic appearances seen. His conclusion is that the lesion is an interstitial neuritis, a syphilitic cirrhosis of vascular

origin and a syphilitic meningitis. The meninges (especially the sub-arachnoid and the pia), are infiltrated with lymphocytes which are specially abundant round vessels; but the vessels which penetrate the nerve are only to a slight extent surrounded by lymphocytes.

In the nerve itself, there is an intense proliferation of vascular connective tissue and of neuroglia, with a subsequent sclerosis and obliteration of the vessels, both pre-existing and new-formed. The atrophy of the nerve fibres depends on the loss of nutrition resulting from the obliteration of the vessels and not on any direct toxic effect on the nerve fibres, and it commences round the vessels, especially in the peripheral subpial zone. According to Léri, the process is primarily a periarteritis and an endarteritis, with subsequent degeneration of nerve tissue. He found the changes in the intracranial portion of the nerve and the chiasma exactly similar to those found in the orbital portion.

The conditions are exactly the same in general paralysis, in tabes, and in other cases of syphilitic atrophy.

Stargardt[43] published his first account of his researches at the Heidelberg Congress in 1911. His full account was published in 1913. He describes the earliest changes as taking place in the intracranial or foraminal portion of the nerve and in the chiasma, and it is only in the fairly late stages that the orbital portion of the nerve, the tract and the external geniculate body show any evidence of change. An exudative process, in which lymphocytes and plasma cells predominate, precedes any evidence of nerve degeneration. This exudation is found mainly in the pial sheath, and the septa spreading in from it. In the pia the plasma cells may be diffusely scattered, but as a rule, they are thickest in the neighbourhood of the vessels. In the septa the plasma cells mostly lie in the Virchow-Robin spaces, but they may permeate all the connective tissue spaces. Not only the larger vessels, but also the capillaries, show plasma cells between the elastic membrane and the thin adventitial layer, and it is along the vessels that they make their way into the nerve. The subsequent degeneration of the myelin sheath and axis cylinder process seems to progress contemporaneously. The disappearance of the degeneration products is due to phagocytic action of cells of glial origin.

His work is summarized briefly in the following paragraphs:—

1. There is no fibre degeneration unless there is an exudative process in some part of the course of the nerve. The exudative process belongs just as much to the picture of optic atrophy as to tabes and general paralysis.

2. The chief sites of the exudative process are the intracranial and foraminal segments of the nerve, and after them the chiasma.

The orbital portion, the tract and the corpus geniculatum are only rarely affected.

3. There is no regularity in the localization and spread of the exudative process.

4. In general paralysis, the exudative process generally spreads from the brain to the optic tracts, etc. In tabes it is an isolated process in the optic nerves or chiasma and may spread to the brain. " All sorts of variations are possible." The exudative process precedes the degenerative changes.

" It is only when distinct degeneration shows in the chiasma that evidences of degeneration in the optic nerve and retina become manifest. The cell degeneration is to be regarded as entirely secondary."

His conclusion is that " the simple optic atrophy in tabes and general paralysis is the result of an extraordinarily chronic inflammation which commences in the neighbourhood of the chiasma, the tract and the intracranial portion of the optic nerve, and makes its way along the lymph sheaths of the vessels into these tissues."

Richter[38] finds himself, as the result of an examination of three cases of tabes and one of tabo-paresis, in complete agreement with Stargardt. The changes in the optic nerve are the same in tabes and tabo-paresis, so that, as far as optic atrophy is concerned, the granulation tissue no longer plays the predominant rôle that he ascribes to it in other tabetic processes. It is an exudative process in which cells of haematogenous origin are predominant, and in this respect the changes are more closely allied to those found in general paralysis than to those found in other tabetic manifestations. Is this a reason for regarding cases in which optic atrophy occurs as really belonging to the category of general paralysis? Mott[22] speaks of the greater tendency of cases with early optic atrophy to pass on to general paralysis; but Richter thinks that the different reaction of the optic nerve can be explained on other grounds. The optic and olfactory nerves differ from all the other nerves of the body. They are of purely ectodermal origin, and their parenchyma is ectodermal in its constitution. Only the septa grow from the sheath into the nerve substance. The more intimate supporting structure of the optic nerve is of glial origin. In the other nerves it is of mesodermal origin. The granulation tissue—Nageotte's " *infiltration embryonnaire* "—is of mesodermal origin and can spread freely in tissues built up on a mesodermal framework, but can find no entry into ectodermal structures. In posterior root lesions the primary changes are in the tissue formed from the combined dural-arachnoid sheaths, and the pia is not represented. In the optic nerve the primary changes are in the pial zone, and the dura and arachnoid sheaths show only scanty signs of granulation tissue. The pia is essentially the

vascular member of the meninges, and when the lesion takes its origin in this the reaction shows a vascular character. In the optic nerve, the primary lesion and its cause, the spirochaetes, have their site in the pia, and in consequence of this, the reaction is of a vascular nature with cells of haematogenous origin.

Summary. Pathological Conclusion

The old hypothesis of a metaluetic toxin, the result of tissue degenerations produced by earlier syphilitic manifestations, must, in light of all the recent work, be finally abandoned. The toxin was always a purely hypothetical substance evoked to explain degenerations taking place where no active organisms could be found. In the light of the work of Noguchi, Levaditi, Mott, Head, Fearnsides, McIntosh and Fildes, it must now be allowed that all the manifestations of syphilis are due to the local production of toxins in the presence of the spirochaete, but that the reaction between the spirochaetes and the tissue varies at different periods and in different tissues, either because of a diminution in the number and virulence of the spirochaetes, or because of differences in the resisting power of the tissues to the action of the toxin. Further, this tissue resistance may vary from time to time owing to variations in the strain on it (Ehrlich) or to the establishment of a condition of *allergie* (von Pirquet).

There still remains, however, the unsettled question as to whether this locally produced toxin acts directly on nerve tissue, producing a parenchymatous degeneration, or primarily on connective, vascular and lymphatic tissues, with a consequent secondary nerve degeneration; or a third possibility, that the nerve degenerations and the connective tissue degenerations are co-ordinate results due to the presence of the spirochaetes in both tissues.

I have failed to find in the course of my reading any strong advocacy of this last hypothesis. Yet I believe there is much to be said for it, and it would bring the causation of optic atrophy into line with the causation of general paralysis as described by McIntosh and Fildes, and it would also be in agreement with the hypothesis as to the origin of tabes which Schaffer[40] very strongly supports. As will be evident from the next part of my paper, I think the clinical evidence tends rather to support this hypothesis.

Functional Disturbances in Tabetic Optic Atrophy

The variability of the functional disturbances found in tabetic optic atrophy is a feature of the condition which has been frequently remarked on. This variability shows itself in the relationship that exists between visual acuity, visual fields and ophthalmoscopic appearances, as well as in the progress of the

disease. Recently the work of Behr[1] has brought into prominence another important functional disturbance, the loss of dark adaptation. This he regards as a most important early symptom of a tabetic atrophy, and he says it may precede any evidence of atrophic change in the fundus. In tabes "there appears regularly a typical and marked loss of dark adaptation, often without the smallest obvious change in the visual acuity, the visual fields or the colour sense." . . . . "A tabetic optic atrophy is always associated with a disturbance of dark adaptation." Uhthoff's[45] dictum that "every visual disturbance in tabes which precedes the ophthalmoscopic signs of optic atrophy is to be regarded as probably unconnected with the progressive optic atrophy," must then be definitely modified. Apart altogether from Behr's work, I consider that Uhthoff's statement is much too sweeping.

Behr divides the clinical course of tabetic atrophy into four stages :—

1*st Stage*.—Isolated disturbance of adaptation with *normal* ophthalmoscopic appearances, visual acuity, visual fields and colour sense.

2*nd Stage*.—Disturbance of adaptation with definite signs of atrophy, but other functions normal.

3*rd Stage*.—Disturbance of adaptation, optic atrophy, loss of visual fields for white and colours, and diminution of visual acuity.

4*th Stage*.—Optic atrophy and amaurosis.

It is outside my brief to discuss the various theories of dark adaptation, but it would seem, if Behr is correct, that the centrifugal fibres are concerned in dark adapation, and that, for some reason or another, they are specially liable to undergo early degenerative change in the course of tabes.

A rarer phenomenon, which is possibly also associated with changes in the centrifugal fibres, is the occasional development of coloured vision. It is mostly described as a blue-green or purple coloration, but Doyne has recently brought forward instances of red coloration in cases of tabes, and Uhthoff[45] mentions a case in which there was a golden shimmer over things on waking in the morning, later turning into a silver shimmer. Eight weeks later the patient complained that in the dark he saw red, and in daylight a blinding whiteness.

Furthermore, it is probable, as a consequence of the diminution of the power of functional adaptation, that some of these patients see much better in diffuse light than in full daylight. When taking the fields of some tabetics, it is very noticeable that on testing along any radius the patient may see the test object very much further out at some times than at others. In most patients, the limits are quite defined, but every now and again

one comes across a patient who gives most variable responses and one cannot get any certain field. This, again, is probably due to disturbance of functional adaptation.

The Visual Fields

In discussing the visual fields of tabetic atrophy, Uhthoff distinguishes between two main classes of defect:—

1. The whole field seems to suffer simultaneously with an early loss of colour fields and early loss of visual acuity. There is a relative peripheral contraction of the fields for white, but a full field may still be obtained in the course of an ordinary investigation, *e.g.*, with 10 mm. white at half metre distance.

2. The area of defective visual fields is sharply delimited from unaffected areas, showing full normal vision, and central acuity may be quite good.

Stargardt gives a more elaborate classification of the visual fields:—

1. Peripheral loss of white and colours with simultaneous loss of function in other parts of the field. With advancing peripheral loss, the visual acuity falls and at the same time, the distinction between red and green, and later blue and yellow.

2. Peripheral loss for white and colours with good function in the untouched fields.

3. Peripheral loss of colours with full fields for white. Visual acuity may be normal or diminished.

4. Partial (sectorial) loss with more or less perfect functioning in the rest of the field.

5. Normal limits to the peripheral fields with diminished visual acuity and colour sensation.

6. Central scotomata.

7. Hemianopic defects.

My first two fields illustrate in the most characteristic way Uhthoff's two classes.

Case I. E.W.M. (age 45), noticed failure of his sight first in January, 1921. He consulted two oculists during 1921, who both told him that nothing could be done for him. Seen February, 1922. Ophthalmoscopically, he showed typical discs of tabetic atrophy. The fields for 10 mm. white test object at 15 inches were quite full, but his visual acuity in each eye was less than 6/60. An attempt to map out colour fields proved quite hopeless as he could see a 2 mm. red or green disc in the most patchy manner over most of the central portion of the field. He would see the colour quite clearly for a moment, and then not at all, and at no time long enough or over a big enough area to allow of my trying to indicate either the outer limits of the area in which colours were visible or the size of the areas in which they were seen.

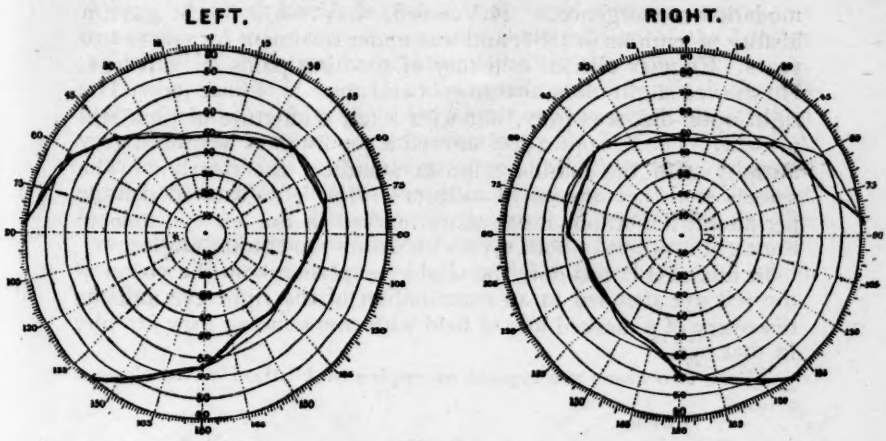


FIG. 2.

L.V. = 6/60. Not Improved. R. V. = 6/60. Not Improved.

CASE I.—E. W. M., Feb. 9, 1922. 10 mm. white at 15 in.

Case II. H.W. (age 46). Seen first in January, 1909. He complained that for some months past he had some difficulty in seeing things with his left eye. His right eye he considered to be quite good. His pupils were unequal—Right more than Left. Neither pupil reacted well to light, but both reacted well on accom-

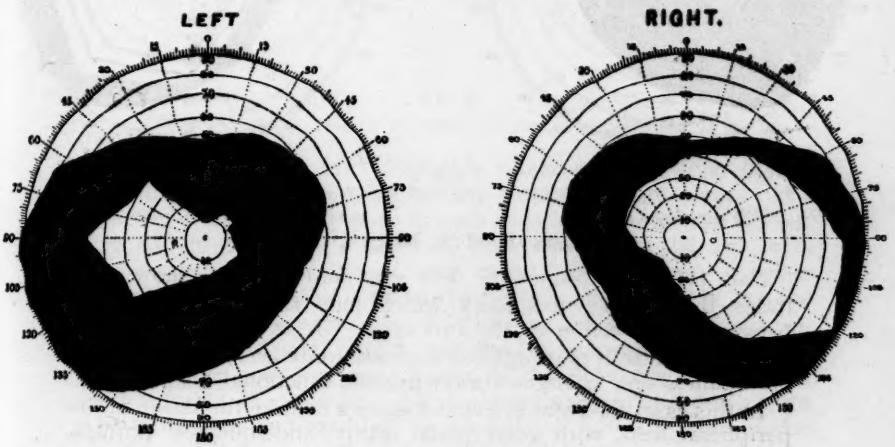


FIG. 3.

L.V. = 6/9. R.V. = 6/5.

CASE II.—H.W., 1909. 10 mm. white at 15 in.

modation-convergence. R.V.=6/5. L.V.=6/9. He gave a history of syphilis in 1887, and was under treatment for one to two years. Enquiry elicited a history of shooting pains in both legs. He showed slight ulnar anaesthesia and absence of knee jerks. His right optic disc was grey, but with a fair admixture of pink still present. The left optic disc showed a condition of advanced grey atrophy with the lamina cribrosa standing out clearly. The vessels were not altered in calibre. His fields showed definite peripheral limitation, much more marked in the left eye than in the right eye, but in both eyes with definite re-entrant angles.

In this case it was only the slight complaint about the vision of the left eye that led to an examination of the right eye and the discovery of a marked loss of field with commencing grey atrophy in that eye.

These two cases are typical examples of Uhthoff's two classes.

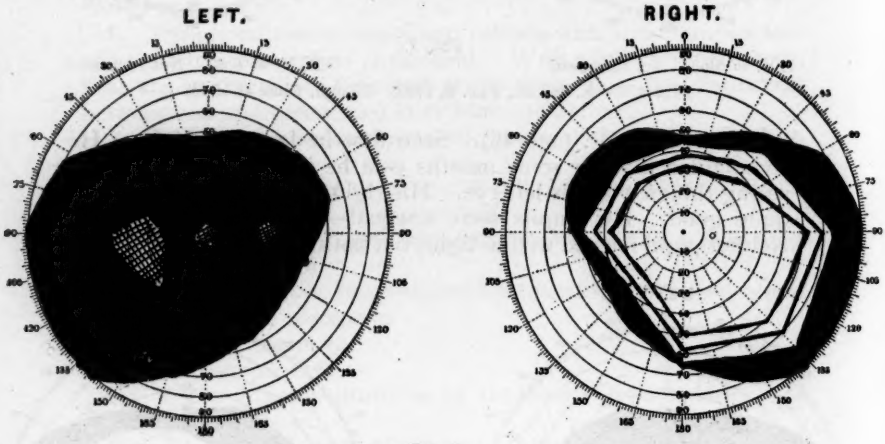


FIG. 4.

L.V. = Fingers, 20 mm. white at 15 in.

R.V. = 6/6. 10 mm., 3 mm., 1 mm. white at 15 in.

CASE III.—T.N., March 4, 1922.

Case III. T.N. (age 39). The next field I have to show illustrates the marked inequality which may sometimes be found between the affection of the two eyes. It is, further, of great interest because it goes against the commonly accepted belief that ophthalmoscopic changes always precede functional disturbance.

In this case, the right eye shows quite a definite limitation of the peripheral field, with good visual acuity and quite a normal-looking disc. The left eye showed a completely atrophic disc, of the usual tabetic type, and the visual field, only obtainable by using a 20 mm. test object, showed only three small islands of

vision, of which one was central, one nasal and the largest one temporal. Again, here it was only the condition of the left eye which called attention to the loss of field in the right eye.

Case IV. McM. (age 51) is one of the cases in which there is quite a good peripheral field for 10 mm. white, but a very marked variability in response to examination with 2 mm. white. A 10 mm. disc had to be used to get a colour field for red, and green could not be seen at all in any part of the field. It is of interest to note that, even allowing for the marked variability in the response for 2 mm. white, there was quite a fair correspondence between the shape of the red colour fields and the 2 mm. white fields.

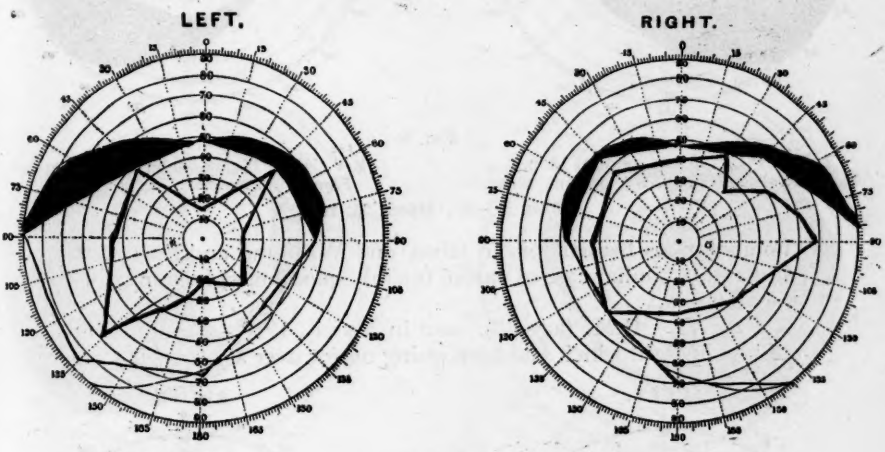


FIG. 5.
CASE IV.—McM., March 25, 1922. 10 mm., 2 mm. white at 15 in.

Case V. F. (age 31). Probably a tabo-paretic, rather than a pure tabes, showed the same marked variability in response. His central vision was very poor in both eyes, and the field in his left eye had to be charted with a 40 mm. disc. In the left eye, examination with a 10 mm. disc showed an indefinite scotoma passing from the blind spot to the macula. In the right eye the field charted with a 10 mm. disc showed its greatest limitation in the upper temporal region. In the left eye the greatest loss is in the upper nasal region. Again, in the right eye examination with a 2 mm. disc shows a scotoma extending up and down from the blind spot and reaching to the fixation point. Colour fields could not be charted. I am afraid it would be stressing this case too far to take it as illustrative of a quadrantic homonymous hemianopia, but Nonne[28] describes a case of partial left-sided

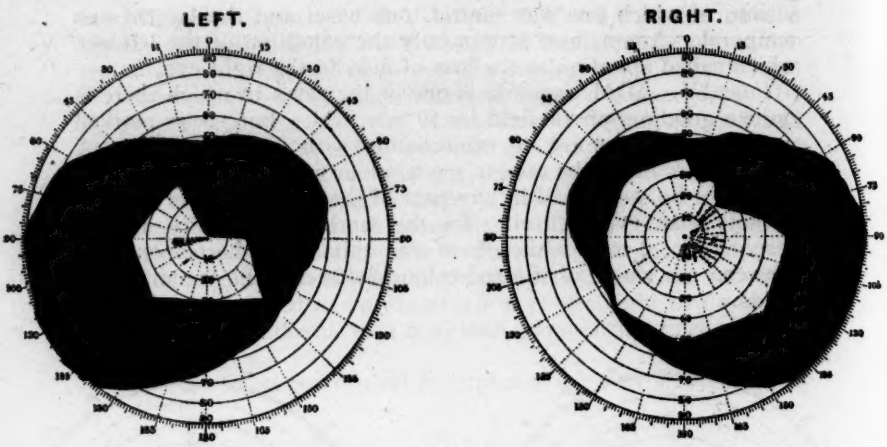


FIG. 6.

L.V. = P.L. 40 mm. white at 15 in. Paracentral scotoma dotted. 10 mm. white.

R.V. 1/60. 10 mm. white. Dotted area. Paracentral scotoma. 2 mm. white.

CASE V.—F., March 26, 1922.

homonymous hemianopia in tabes, and Wilbrand and Saenger[49] illustrate a similar case of partial (mainly quadrantic) homonymous hemianopia.

Case VI. B.M. (age 43), seen in March, 1918. Complaining of loss of sight which had been going on for over a year. He was

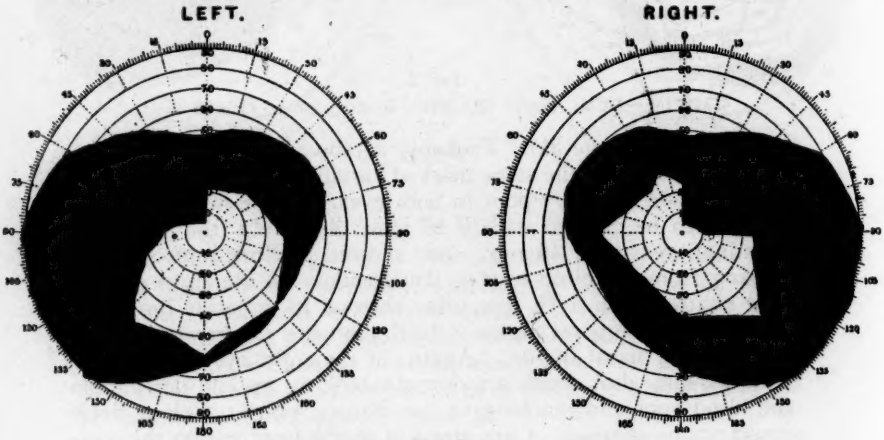


FIG. 7.

L.V. = 6/18.

R.V. = 6/36.

CASE VI.—B.M., March, 1918. 10 mm. white at 15 in. Colours lost.

sent to me with the definite diagnosis of tabes already made. His pupils were quite inactive to light, but reacted with accommodation-convergence. His corrected vision in the right eye was 6/36, and in the left eye 6/18. He complained that he saw worse in bright light than in diffuse daylight. His discs were very atrophic, but the blood vessels were of quite good calibre. His visual fields showed such a marked bitemporal quadrantic loss that I determined to ascertain whether there might not be some pituitary disease complicating the tabes, but the radiograph showed a perfectly normal fossa. His subsequent history shows a steady but slow advance, and he is still able to manage a large farm, though his R.V.=1/60, and L.V.=5/60 only.

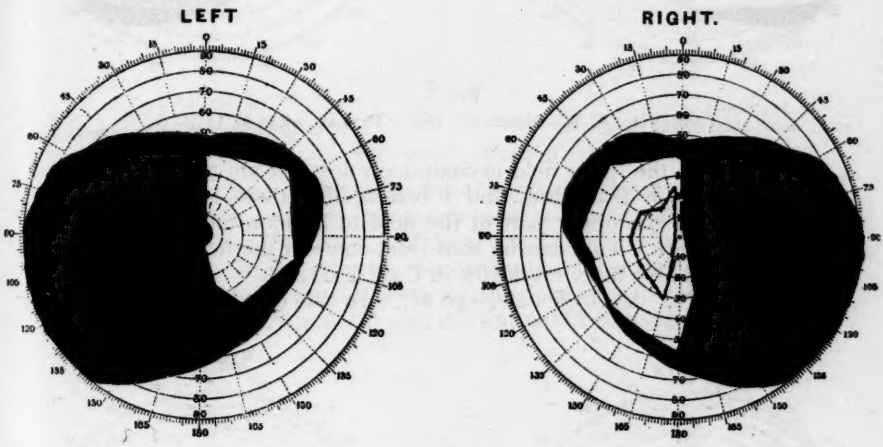


FIG. 8

Wilbrand and Saenger, "*Neurologie des Auges*," Vol. V, p. 553.

In connection with this case, I illustrate the fields of a case cited by Wilbrand and Saenger[49] (*Neur. des Auges*, Vol. V, p. 553) which shows an absolute bitemporal hemianopia in a tabetic.

Case VII. M.W. (female, age 52). I cite this case for the special reason that the marked nasal quadrantic loss and the atrophy of the disc led to her undergoing an operation for chronic glaucoma in the right eye. Apart from this, there is nothing especially noteworthy about the case.

Uhthoff[45], in his article in Graefe-Saemisch, Vol. XI, gives an example of a purely horizontal hemianopic loss in a case of tabes. In that case the upper part of the field in both eyes is hardly

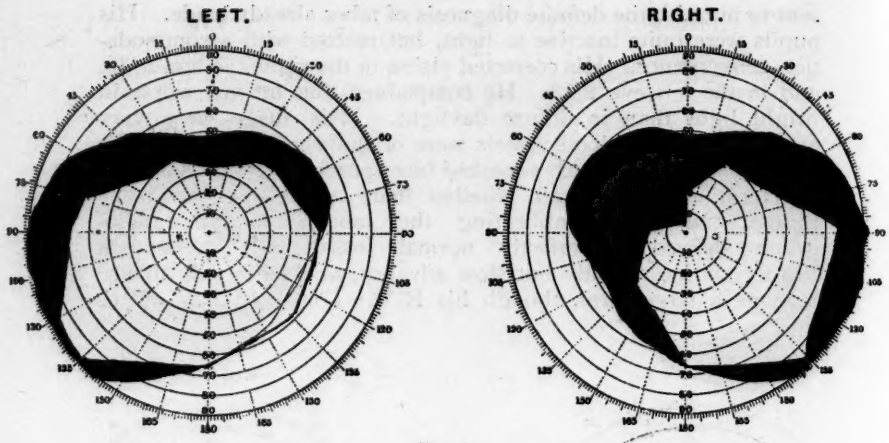


FIG. 9.

CASE VII. M.W., March 20, 1922. 10 m.m. white at 15 in.

affected, but the lower field is completely lost. I am afraid I can cite no case of that kind, and I believe that there is a greater tendency for the upper part of the field to be more affected than the lower part. That can be seen from some of the fields already shown, and also more markedly in Case VIII.

Case VIII. M.C. (female, age 42). In this case, the right eye

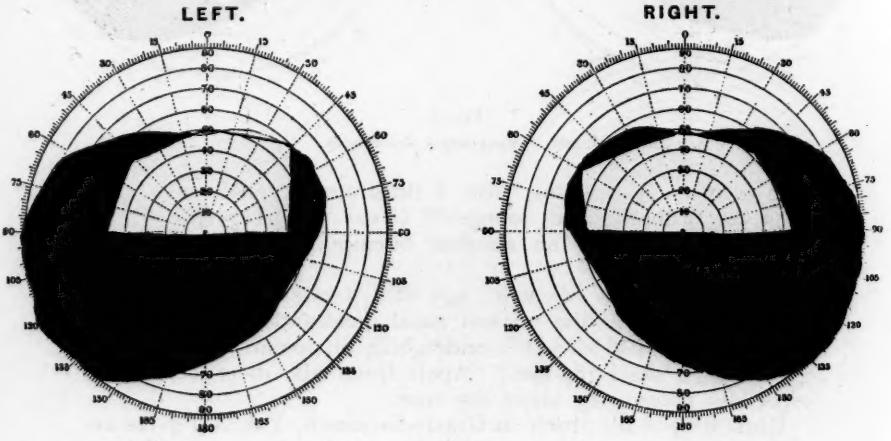


FIG. 10.

Uhthoff, Graefe-Saemisch, *loc. cit.* p. 198.

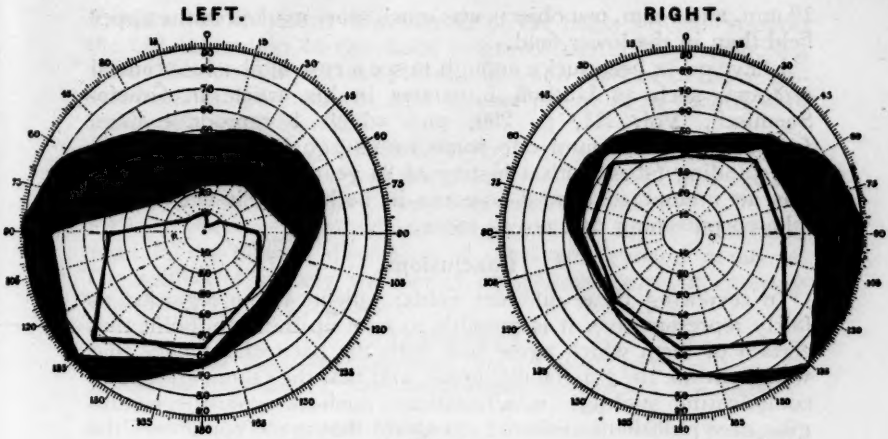


FIG. 11.

L.V.=6/12.
10 mm. and 2 mm. white at 15 in.

R.V. Amblyopic eye.
V.=6/60. 10 mm. and 2 mm. white

CASE VIII.—M.C., March 27, 1922.

had always been amblyopic, and though the disc was pale, it did not show the ordinary appearances of a tabetic atrophy. The vision was less than 6/60, and there was a general slight peripheral limitation. In the left eye, the ophthalmoscope showed a typical tabetic atrophy of the disc with bluish-white appearance and sharp-cut outline. L.V.=6/12, and the limitation of the field, both for

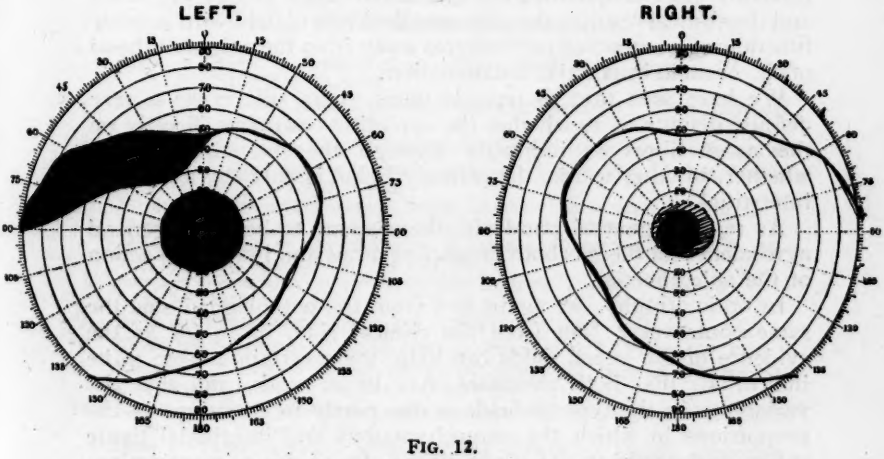


FIG. 12.

Uhthoff, Graefe-Saemisch, "Handbuch d. ges. Augenheilk," Vol. XI, p. 200

10 mm. and 2 mm. test objects was much more marked in the upper field than in the lower field.

I have never been lucky enough to see a case of absolute central scotoma, such as Uhthoff illustrates in his article in Graefe-Saemisch, Vol. XI, p. 200, and which I reproduce here. I thought I had found one some weeks ago in a case of very old standing tabes (with a history of 25 years), but though there was an undoubted central scotoma in both eyes, it was due to tobacco poisoning and not to tabes.

Conclusions

In reviewing these different fields, which can be regarded as fairly representative, it is possible to take up the standpoint that certain of them which agree best with the particular theory that we favour are the true tabetic fields, and that the others are due to complicating syphilitic manifestations, such as syphilitic meningitis or syphilitic myelitis. I am afraid that is too commonly the standpoint of writers on the subject. The other explanation, which seems to me the more probable one, is that the variability in the fields is a true exemplification of the variability in the pathological process; that in tabetic optic atrophy we have a process in which both parenchymatous tissue and interstitial tissue are attacked by the toxin. In some cases the parenchyma suffers more severely and at an earlier stage, and then there is presented the rapid diffuse loss of function involving the central acuity, the functional adaptation and the colour fields, but often leaving an almost full field to larger white objects. In other cases, and probably more frequently, the interstitial tissue suffers the most, and the clinical result is the circumscribed loss of field with actively functioning unattacked parenchyma away from the neighbourhood of the chronic interstitial inflammation.

We have seen that as regards tabes, there still exists a very definite dispute as to whether the syphilitic toxin acts directly on the nerve fibres or indirectly through the interstitial tissue, whether, in other words, the primary lesion is parenchymatous or interstitial.

As regards general paralysis, there seems to be more general agreement that there is both parenchymatous and interstitial action of the spirochaetes.

In optic atrophy, we again find from the pathological side the same controversy; but from the clinical side, in so far as the evidence of the visual fields can help, there certainly seem to be indications that both processes may be at work, and that the variability in the type of fields is due partly to variations in the proportions in which the parenchymatous and interstitial tissue suffer, and partly to variations in the site of the primary lesion.

Further, an unbiassed examination of the fields will not support the old contention of the distal origin of the tabetic lesion, but shows that, in many cases at least, the lesion must be in the neighbourhood of the chiasma, and to that extent is in accord with Stargardt's contentions.

It will always be a matter of regret that pathological work so valuable as Stargardt's was not supported by visual fields of the cases which subsequently came under examination.

I have purposely omitted any mention of the Argyll Robertson pupil in the general discussion of tabes and general paralysis. The subject is already sufficiently complicated without entering into that very dark region.

REFERENCES

1. **Behr.**—*Klin. Monatsbl. f. Augenheilk*, Vol. LV, Part ii, 1915.
2. **Cassirer and Strauss.**—"Tabes dorsalis incipiens und Syphilis." *Monatsschr. f. Psychiatrie u. Neurol.*, Vol. X, part iv, p. 241, 1901.
3. **Charcot.**—"De l'amaurose tabétique." *Mouvement médical*, November; *Jl. d'Ophtal.*, Vol. I, pp. 612-617, 1872.
4. **Dejerine.**—"Contribution à l'étude de l'ataxie locomotrice des membres supérieurs." *Arch. de Physiol. norm. et pathol.*, 4 Ser. Vol. I, p. 331, 1888.
   Idem.—"Maladie de la moelle epinière." Paris, 1909.
5. **Dejerine and J. Martin.**—"De l'atrophie des nerfs optiques dans le prognostic de la sclérose des cordons postérieurs." *Compt. rend. hebt. des Séances de la Soc. de Biol.*, No. 25, p. 431, 1889.
6. **Elschnig.**—"Zur Anatomie der Sehnervenatrophie bei Erkrankungen des Centralnervensystems." *Wiener Klin. Wochenschr.*, 12 Jahrg., No. 11, p. 275, 1899.
7. **Erb.**—"Zur Pathologie der Tabes dorsalis." *Deut. Arch. f. klin. Med.*, Vol. XXIV, p. 1, 1879.
   Idem.—"Zur Aetiologie der Tabes dorsalis." *Berliner Klin. Wochenschr.*, Vol. XX, p. 481, 1883.
   Idem.—"Zur Aetiologie der Tabes." *Berliner Klin. Wochenschr.*, Vol. XXVIII, pp. 713 and 751, 1891.
   Idem.—"Syphilis und Tabes." *Berliner Klin. Wochenschr.*, Vol. XXIX, p. 554, 1892.
   Idem.—"Tabes." *Festschrift der Deut. Zeitschr. f. Nervenheilk.*, Vol. XLVII-XLVIII, p. 74, 1913.
8. **Flechsig.**—"Ist die Tabes dorsalis eine Systemerkrankung?" *Neur. Centralbl*, Vol. IX, pp. 33 and 72, 1890.
9. **Gliksmann.**—"Ueber Sehnervenatrophie bei Tabes." Inaug. Diss. Freiburg, 1900.
10. **Gowers.**—"Axial Neuritis in Spinal Disease." (Ophth. Soc. of Great Britain.) *Brit. Med. Jl.*, Vol II, p. 932.
    Idem.—"Unilateral Hemiopia in Spinal Atrophy." *Brit. Med. Jl.*, Vol. II.
    Idem.—"Syphilis and Locomotor Ataxy." *Lancet*, Vol. I, p. 94, 1881.
    Idem.—"Address on Eye Symptoms in Diseases of the Spinal Cord." *Med. Times and Gazette*, Vol. I, p. 661, 1883.
    Idem.—"Memoranda on Eye Symptoms in Spinal Disease." *Lancet*, Vol. I, p. 869, 1883.
11. **Head and Fearnsides.**—*Brain*, Vol. XXXVII, p. 1, 1914.
12. **Kaufmann.**—*Zeitschr. f. d. ges. Neur. u. Psych.*, Vol. LXVII, p. 190, 1921.
13. **Klippel.**—"L'atrophie du nerf optique par rapport au tabes et à la paralysie générale." *Rev. de Psychiatrie*, April, No. 4.
14. **Köster.**—"Zur Physiologie f. Spinal-ganglienzellen, u.s.w., sowie zur Pathogenese der Tabes." Leipzig, 1904.
    Idem.—"Tabes dorsalis; ophthalmoplegia externa." *Upsala läkareförens förhandl.*, No. 9, p. 126.

15. **Leber.**—"Beiträge zur Kenntnis der atrophischen Veränderungen des Sehnerven." *Arch. f. Ophthal.*, Vol. XIV, Part ii, p. 164, 1868.
Idem.—"Die Krankheiten der Netzhaut und des Sehnerven." Handb. d. ges. Augenheilk. v. Graefe-Saemisch, Vol. V, Kap. viii, p. 838, 1877.
16. **Léri.**—*Nouvell. Iconogr. de la Sa'pêtrière*, Vol. XVII, Nr. 5, p. 304, 1904.
17. **Marie et Léri.**—*Revue neurolog.*, Vol. XIII, p. 246, 1905.
18. **Marie.**—"Contribution à l'étude des troubles oculaires dans la paralysie générale." Thèse de Paris, 1890.
19. **Marina.**—"Zur Symptomatologie der Tabes dorsalis mit besonderer Rücksicht auf Ohren, Kehl- und Schlundkopf." *Arch. f. Psych. u. Nervenkr.*, Vol. XXI, p. 156, 1890.
20. **Marinesco.**—"Contrib. à l'étude de l'histologie et de la pathogenie du Tabes." *Semaine méd.*, p. 181, 1906.
21. **McIntosh and Fildes.**—*Brain*, Vol. XXXVI, p. 1, 1913.
22. **Mott.**—"A System of Syphilis." Vol. IV. Oxford, 1910
23. **Moxter.**—"Beitrag zur Auffassung der Tabes als Neuronerkrankung." *Zeitschr. f. klin. Mediz.*, Vol. XXIX, p. 334, 1896.
24. **Nageotte.**—"Tabes et paralysie générale." Thèse de Paris, 1893.
Idem.—La lésion primitive du tabes." *Bull. de la Soc. de Biol.*, 10 Ser., Vol. I, p. 713, 1894.
Idem.—"Etude sur la meningomyelite diffuse dans le tabes, la paralysie générale et la syphilis spinale." *Arch. de Neurol.*, Vol. XXX, p. 273, 1895.
Idem.—"Etude sur un cas de tabes uniradiculaire chez un paralytique général." *Rev. Neurol.*, 1895.
Idem.—"Note sur la présence de fibres à myéline dans la pie-mère spinale des tabétiques, en rapport avec la régénération de fibres radiculaires antérieures." *Compt. rend. de la Soc. de Biol.*, p. 738, 1899.
Idem.—"Note sur la lésion primitive du tabes." *Compt rend. de la Soc. de Biol.*, p. 354, 1900.
Idem.—"Note sur les formations cavitaires par périnévrite dans les nerfs radiculaires." *Extrait des Compt. rend. de la Soc. de Biol.*, 11 Ser. IV, p. 1443, 1902.
Idem.—"Note sur les lésions radiculaires et ganglionnaires du tabes." *Compt. rend. de la Soc. de Biol.*, Vol. LIV, p. 1080, 1902
Idem.—"Pathogenie du Tabes dorsal." G. Naud, éditeur, Paris, 1903.
Idem.—"Contribution à l'étude anatomique des cordons postérieurs." *Nouvelle Iconographie de la Salpêtrière*, Vol. XVII, p. 17, 1904.
Idem.—"Régénération collatérale des fibres nerveuses terminées par des massues de croissance, à l'état pathologique et à l'état normal; lésions tabétiques des racines médullaires." *Nouvelle Iconographie de la Salpêtrière*, Vol. XIX, p. 217, 1906.
25. **Noguchi.**—"The Transmission of Treponema Pallidum from the Brains of Paretics to the Rabbit." *Jl. Amer. Med. Assoc.*, Vol. LXI, p. 85, July 12th, 1913.
26. **Noguchi and J. W. Moore.**—"A Demonstration of Treponema Pallidum in the Brain in Cases of General Paralysis." *Jl. Exper. Med.*, Vol. XVII, p. 232, Feb., 1913.
27. **Nissl.**—"Ueber die Veränderungen der Ganglienzellen am Fazialiskern des Kaninchens nach Ausreissung der Nerven." *Allgem. Zeitschr. f. Psych.*, Vol. XLVIII, p. 197, 1892.
Idem.—"Zur Lehre von der Hirnlues." *Neurolog. Zentralbl.*, p. 42, 1904.
28. **Nonne.**—*Neurol. Centr.*, p. 228, 1899.
Idem.—*Berlin. Klin. Wochenschr.*, Nos. 15, 16, 17, 1899.
Idem.—"Zur Kasuistik der Beteiligung der peripherischen Nerven bei Tabes dorsalis." *Arch. f. Pysch. u. Nervenkr.*, Vol. XIX, Part ii, p. 352, 1888.
Idem.—"Syphilis und Nervensystem," 1921.
29. **Obersteiner.**—"Bemerkungen zur tabischen Hinterwurzelerkrankung." Obersteiners Arbeiten, Part iii, 1895.
Idem.—"Die Pathogenese der Tabes." *Berl. klin. Wochenschr.*, No. 42, p. 905, 1897.

30. **Obersteiner and Redlich.**—" Ueber Wesen der Pathogenese der tabischen Hinterstrangsdegeneration." Arb. aus dem hirnanal Inst. Wien, Part ii, p. 158, 1894.
Idem.—" Krankheiten des Rückenmarks." Handbuch der prakt. Med. (W. Ebstein), Stuttgart, Vol. IV.
Idem.—" Ueber Wesen und Pathologie der tabischen Hinterstrangsdegeneration." Arb. aus d. Inst. Obersteiner, Part ii, Wien, 1894.

31. **Oppenheim and Siemerling.**—" Beiträge zur Pathologie der Tabes dorsalis und der peripherischen Nervenerkrankung." *Arch. f. Psych. u. Nervenkr.*, Vol. XVIII, Part i and ii, pp. 98 and 487, 1887.

32. **Oppenheim.**—" Beiträge zur Pathologie der Hirnkrankheiten." *Charité-Analen*, Vol. X, p. 335, 1883.
Idem.—" Beiträge zur Pathologie der Tabes dorsalis." *Arch. f. Psych. u. Nervenkr.*, Vol. XX, p. 131, 1889.
Idem.—" Zur Kenntnis der syphilitischen Erkrankungen des Centralnervensystems." Berlin, 1890.

33. **Orr and Rows.**—*Brain*, Vol. XXXVI, p. 271, 1903.
Idem—Brain, Vol. XXXVII, p. 460, 1904.

34. **Parsons.**—" The Pathology of the Eye," Vol. IV, p. 1344, 1908.

35. **Peltesohn.**—" Ursachen und Verlauf der Sehnervenatrophie." *Centralbl. f. Augenheilk.*, Feb., p. 45; March, p. 75; April, p. 106.

36. **Popow,**—" Beitrag zur Kenntnis der Sehnervenveränderungen bei Tabes dorsalis." *Deutsche. Zeitschr. f. Nervenheilkunde*, Vol. IV, p. 270, 1893.

37. **Redlich.**—" Die hinteren Wurzeln des Ruckenmarkes und die. path. Anat. der Tabes dorsalis." *Jahr. f. Psych.*, No. 11, p. 1, 1892.
Idem.—" Die Pathologie der tabischen Hinterstrangserkrankung." G. Fischer, Jena, 1897.

38. **Richter.**—*Zeitschr. f. die gesamte Neurol. und Psychiatrie*, Vol. LXVII, p. 1, 1921.

39. **Rönne.**—" Rührt die Optikusatrophie durch Tabes von einen Leiden der Ganglienzellen oder der Nervenfasern her." *Arch. f. Ophthal.*, Vol. LXXII, Part iii, p. 481, 1909.

40. **Schaffer** —" Das Verhalten der Spinalganglien bei Tabes auf Grund Nissls Färbung." *Neurol. Centralbl.*, p. 2, 1898.
Idem.— " Ueber Nervenzellveränderungen des Vorderhornes bei Tabes." *Monatsschr. f. Psych. u. Neurol.*, Vol. III, p. 64, 1898.
Idem —" Beiträge zur Histopathogenese der tabischen Hinterstrangsdegeneration." *Dtsch. Zeitschr. f. Nervenheilk.*, Vol. XIII, p. 287, 1898.
Idem.—" Anatomisch-Klinische Vorträge aus dem Gebiete der Nervenpathologie." Jena, 1901.
Idem.—" Ueber Fibrillenbilder tabischer Spinalganglien." *Zeitschr. f. d. ges. Neurol. u. Psychiatr.*, Vol. I, Part iii, p. 439, 1910.
Idem.—" Tabes dorsalis," in Lewandowsky's " Handbuch der Neurologie," Vol. II, 1910.
Idem.—" Die Plasmazellen." Jena. 1910.
Idem.—Zeitschr. f. d. ges. Neurol. u. Psychiatr., Vol. LXVII, p. 222, 1921.

41. **Schlagenhaufer.**—" Anatomische Beiträge zum Faserverlauf in den Sehnerven: bahnen und Beitrag zur tabischen Sehnervenatrophie." *Jahrbuch. f. Psych. u. Neurol.*, Vol. XVI, p. 1, 1897.

42. **Sherrington.**—*Brain*, Vol. XLI, p. 332, 1918.

43. **Stargardt.**—*Bericht Deutsche Oph. Ges.*, Heidelberg, 1911.
Idem.—Biolog. Abt. des ärztl. Vereins in Hamburg, Vol. XXVI, Part xi, 1912.
Idem.—" Zur pathologischen Anatomie des Sehnervenschwundes bei Tabes und progressiver Paralyse." *Bericht über die 38. Vers. der Heidelberger ophthal. Ges.*, p. 214, 1912.
Idem.—Münchener med. Wochenschr., No. 5, p. 269, 1913.
Idem.—" Ueber die Ursachen des Sehnervenschwundes bei der Tabes und der progressiven Paralyse." Aug. Hirschwald, Berlin, 1913.

44. **Stroebe.**—" Ueber Veränderung der Spinalganglien bei Tabes." *Centralbl. f. allg. Pathol. und path. Anat.* Vol. V, p. 853, 1894.

45. **Uhthoff.**—"Beitrag zur Sehnervenatrophie." *Arch. f. Ophthal.*, Vol. XXVI, p. 244, 1880.
Idem.—"Weitere Beiträge zur Sehnervenatrophie, Sehnerv. u. Netzhauterkrankungen bei Allgemeinerkrankung." Verlag von H. Peters, Berlin.
Idem.—Graefe-Saemisch, Vol. XI, 1903.

46. **v. Grosz.**—"Die Atrophie der Optikusnerven bei Tabes dorsalis." *Ber. über den 9 internat. Ophth.-Kongr. in Utrecht*, 1899.

47. **v. Strümpell.**—"Die pathologische Anatomie der Tabes." *Arch. f. Psych.*, Vol. XII, p. 723, 1882.
Idem.—*Neurol. Centralbl.*, Vol. VII, p. 122, 1888.
Idem.—"Beiträge zur Pathologie des Rückenmarks." *Arch. f. Psych. u. Nervenkr.*, Vol. XI, pp. 27 and 723.
Idem.—"Lehrbuch der speziellen Pathologie und Therapie." Leipzig, 17th Aufl., 1909.

48. **Wagenmann.**—"Schwund markhaltiger Nervenfasern in der Retina in Folge von genuiner Sehnervenatrophie bei Tabes dorsalis." *Arch. f. Ophth.*, Vol. XL, p. 256, 1894.

49. **Wilbrand and Saenger.**—"Neurologie des Auges," Vol. V, 1913.

BUTYN: A SUBSTITUTE FOR COCAIN

BY

W. M. BEAUMONT

BATH

THE pre-eminence of cocain as a local anaesthetic is challenged by butyn. This synthetic product of coal-tar was introduced by the Abbott Laboratories of Chicago for surface anaesthesia, especially in ocular, nasal, pharyngeal and dental diseases. Its chemical formula is para-aminobenzoyl gamma di-n-butylamino-propanol sulphate.

$$[NH_2\,C_6\,H_4\,COO\,(CH_2),\,N\,(C_4H_9)_2]_2\,H_2,\,SO_4.$$

The chemical research work was done by Profs. Roger Adams and Oliver Kamm, of the University of Illinois, and Dr. E. H. Volveiller, of the Abbott Laboratories.

Butyn has been reported on by a committee on local anaesthesia appointed by the section of Ophthalmology of the American Medical Association. The members were:—Albert E. Bulson, Jun., Fort Wayne (Chairman); Wm. Zentmayer, Philadelphia; Roger S. Thomson, New York City; H. Maxwell Langdon, Philadelphia; Harry S. Gradle, Chicago.

Their report* shows that one minute after one instillation of 2 per cent. solution surface anaesthesia is sufficient to permit the removal of foreign bodies from the cornea without discomfort. Anaesthesia lasts from 15 to 20 minutes, or it may be 30 minutes. The depth of anaesthesia is not sufficient by one instillation for operations or for removal of *deeply* imbedded foreign bodies in the cornea, but it is sufficient for the application of astringents and

* *Journal of the American Medical Association*, February 4, 1922, p. 343.

for the determination of intraocular pressure with the tonometer. When a number of instillations have been made there is marked increase in the depth, duration and degree of the anaesthesia. For operative work (as with cocain) four instillations, 3 minutes apart, are sufficient. Operative work is to be begun from 5 to 10 minutes after the last instillation. The anaesthesia which results is sufficient for all the commoner major operations except enucleation, which has not been tried. The height of anaesthesia is reached in 5 to 8 minutes after the fourth instillation, and its duration lasts from 20 to 30 minutes, or even nearly an hour.

Side actions.—One instillation almost always produces a mild hyperaemia of the conjunctiva, not increased by subsequent instillations. It is controlled by epinephrin solution.

Butyn does not affect the pupil diameter, nor increase the ocular pressure. There is no desiccation or disturbance of nutrition of the cornea. A solution of butyn does not deteriorate rapidly when exposed to the air and light, nor is it impaired by boiling. In no instance, including hundreds of times that butyn has been used by members of the Committee, for minor and major eye operations, as well as in operative work in the nose and throat, have the slightest systemic toxic manifestations been noted. It has also been used for surface and infiltration anaesthesia, and in paste for topical application in nose and eye work, without toxic effect.

" Our Committee is unanimous in the opinion that butyn for surface anaesthesia for minor operations is superior to cocain." It acts more quickly, fewer applications are required, there is no dilatation of the pupil or desiccation of the cornea and anaesthesia is more profound. For iridectomy and cataract extraction there is a more profound anaesthesia than occurs with cocain. For operations on the extrinsic muscles the results are equal to cocain, although the Committee believes that a stronger solution than 2 per cent. may be preferable. For infiltration anaesthesia, using both a 0.50 and 1 per cent. solution, the Committee has had " very satisfactory results."

Conclusions :—

(1.) Butyn is more powerful than cocain, a smaller quantity being required.

(2.) It acts more rapidly than cocain.

(3.) Its action is more prolonged.

(4.) It is less toxic.

(5). There is no drying effect on the tissues.

(6.) There is no change in the size of the pupil.

(7.) There is no ischaemic effect and therefore no shrinking effect on the tissues.

8. It can be boiled without impairing its efficiency.

Butyn compared with other local anaesthetics. Butyn is 2½ times more toxic than cocain when injected hypodermically into albino rats, but in cats the effects are about equal.

In a preliminary report of the Council on Pharmacy and Chemistry of the American Medical Association,* it is stated that on the normal human eye a 0.50 per cent. solution of butyn is less efficient than a 1 per cent. solution of holocain, but more efficient than a 1 per cent. solution of cocain, or a 1 per cent, solution of eucain. Butyn solutions are non-irritant. Like alypin, butyn is not mydriatic, and it does not affect the accommodation. It may take the place of cocain for surface anaesthesia of mucous membranes. It is superior for use in the eye to other synthetic anaesthetics for the reason that it can be used in lower concentrations (presumably because of more prompt absorption).

On the other hand butyn is not promising for injection or spinal anaesthesia since its toxicity is greater than that of procain.

The following table shows the comparative efficiency of butyn compared with procain and cocain :—

	Efficiency on Motor Nerves.	Efficiency on Sensory Nerve Trunks.	Efficiency on Rabbits' Cornea	Efficiency on Frogs' Skin.
Cocain	1	1	1	1
Procain ...	1	½	⅓	⅛
Butyn	8	2	1	2

Butyn in dentistry has been used in the United States for extractions both by conduction and by infiltration methods. In the latter 1 c.c. of a 0.5 per cent. solution has been injected without producing toxic symptoms. But as its use by dentists is still in the experimental stage it is recommended that it should be used cautiously. The aphrodisiac effects of cocain which are sometimes noticeable after injections of the drug to produce local anaesthesia in dental practice† have not been recorded after the use of butyn.

My own limited experience of butyn confirms the clinical findings of the Committee appointed by the American Medical Association.

I have used it in 2 per cent. solution for the extraction of cataract and for iridectomy, with satisfactory results.

For the removal of foreign bodies in the cornea and conjunctiva

* *Journal of the American Medical Association*, December 10, 1921, p. 1,891.
† *British Medical Journal*, May 27, 1922.

1 per cent. solution is usually sufficient to produce anaesthesia without causing conjunctival injection and there is never any mydriasis.

In the treatment of glaucoma if butyn is combined with eserin there is seldom any complaint of twitching or discomfort as is sometimes the case when eserin is used alone.

For all the everyday purposes for which cocain is used in ophthalmic practice butyn appears to be an ideal alternative and one which is devoid of the disadvantages of mydriasis and desiccation of the cornea. When used with other medicaments it is necessary to remember that butyn is incompatible with chlorides.

I have also used butyn as a lotion to relieve pain in herpes of the head and eye and in detachment of the retina.

In view of the widespread illegal administration of cocain, the potentialities of a substitute, devoid of attractions for the drugtaker, should be of value, and if further investigation confirms all that is claimed for butyn, we may hope that the results will have far-reaching effects in reducing the manufacture and repressing the distribution of a drug the illicit use of which causes social calamities.

THE VISION OF RAILWAYMEN

BY

LEONARD J. C. MITCHELL, M.B., B.S. (Melb.)

ASSISTANT EXAMINER IN VISION, ETC., VICTORIAN RAILWAYS

IN order to arrive at some more definite relation between vision as tested by test types under good conditions, and what may be termed " safe vision " from a railway point of view, a series of tests was undertaken recently (1920) at Tottenham (Vic.). Previously (1898) Barrett, Orr and Murray had conducted similar tests, but their data were too meagre to give any definite result, and further, the main object before these experimenters was to prove that what is called a " practical test " is, by itself, very misleading. Later (1914) Gault, Webster and Ryan, in collaboration with Stanley and the writer, conducted a test with a special signal, but it would seem that fallacy was introduced by first reducing with glasses the vision of persons examined. The New South Wales Railway Medical Officers have recorded a test in which the persons examined had vision below normal, but the fallacy of their test was that there was a different background for the various signals used. Bearing in mind these unsatisfactory results, the Tottenham experiment was planned and carried out by H. R. Stanley and the writer, on the afternoon of a cloudy calm

day, a special standard signal was erected, and the sky was the background throughout the tests.

A standard signal arm (Vic. Rlys.) measures 4 ft. 9 in. by 10 in., and this arm subtends an angle of five minutes at a distance of 1,085 yards. It might be inferred from Snellen's figures that such a signal arm would be read accurately by a person with 6/6 vision at that distance and no further, but the signal can be read by a normal-sighted person up to a distance of 1.5 miles. Testing vision by test types implies a good illumination, and if we are to form an opinion as to the relation between form vision as tested in the consulting room, and the ability to read signals accurately, it is necessary to test that ability under good conditions of illumination and background. Further, it must be remembered that normal vision is, as a rule, very much better than 6/6, probably 6/4 would be nearer the mark among a large number of normal sighted, and so the results of the test described below are not as surprising as would first appear.

The subjects for examination were locomotive drivers, signalmen and station masters, and each man's vision was tested with test types under the best indoor conditions immediately before the outdoor test was begun. One man, with vision R. 6/12, L. 6/12, combined 6/9, read the signal accurately at 1,760 yards, two others with the same vision were accurate at 1,600 yards, and another at 1,500 yards, men with vision R. 6/18, L. 6/18, combined 6/12, were accurate at from 1,500 to 1,600 yards.

The variations in the ability to read signals amongst men whose vision was the same as when tested indoors, was undoubtedly due to the nature of the error in the individual cases, that is, whether the error was astigmatism or simple hypermetropia. (A temporary light smoke haze obscured the view at one stage, but it was shown that a different visibility of as much as 300 yards was made.)

It is, therefore, clear that we have here a definite relation between the vision of a man whose best vision is 6/12 with both eyes open as tested indoors, and the distance at which he can read signals under good conditions. In other words, a relation between certain degrees of defective vision, and the practical value of such vision from a railway point of view, has been established.

As a matter of railway practice, the signals are so arranged as to come into view at 600 yards distance as a minimum. (This does not apply to automatic signals.) So it would appear that there is an ample margin of safety as regards the distance at which a man with 6/18, 6/18, 6/12 vision can see signals.

With regard to the seeing of the signals at night, the lamp glass of a standard signal (Vic. Rlys.) is 5 in. in diameter, but the candle power of the lamps depends much on the cleanliness of the glass, etc. As a point of light is very much more readily seen than the

form of a letter, the size of the lamp glass does not bear the same relation to the distance at which the lamp is visible, as in the case of form vision.

The same men were tested with the Edridge-Green lantern immediately after the outdoor test, and it was shown that each man with vision 6/18, 6/18, both 6/12, was quite accurate with dull red lights. This is our everyday experience, and we hold that if the form vision is not reduced below 6/12 with both eyes open, the colour perception is safe—always excepting toxic amblyopia. A shortened red end to the spectrum should not be diagnosed unless the vision is improved to 6/6, or unless a large (half inch) aperture be used.

Conclusions :—

(1.) Under ideal conditions a man with vision R. 6/18, L. 6/18, both 6/12, can, with both eyes open, read signals accurately at 1,500 yards.

(2.) Provided that the vision with both eyes open is not below 6/12, colour *perception is not dangerously lowered.*

NOTE.—The complete records of these experiments are in the files of the Railway Department referred to.

REMOVAL OF FILARIA FROM UNDER THE CONJUNCTIVA

BY

E. CHARLES

GUJRANWALA

BAGGA S/O HIRA, aged 45, resident of Sialkot District, near Jammu State Territory, had, some eight months ago, sudden severe pain in his right eye, and soon after he felt a little swelling on the inner and downward side of his eyeball. The pain of a neuralgic type stopped when cold, but increased in the daytime.

Present condition.—Fairly large swelling on the inner and downward side of the right eye beneath the ocular conjunctiva, redness, watering, pupil dilated, patient in great pain, vision not affected.

Operation.—Under cocain, two days after admission, a small incision with a Graefe's knife was made in the swelling, and a loop of the living worm began to come out of the opening. Just when the head of the thing was being pulled at the patient complained of severe pain, the pull was loosened and the worm began to crawl in again ; when just a little of the worm had gone back a sudden pull

brought out the whole of the worm. Recovery was uneventful, the patient was discharged cured about a week after. I should be glad to hear through your paper if anyone else has ever had any worm like that removed from an eye, and something more about the mode of its location in the eye.

Note on Mr. Charles's Specimen by Major Sewell

The specimen by Mr. Charles is unfortunately so contracted and shrunken, owing to its having been preserved in spirit, that it is impossible to arrive at any definite conclusion with regard to it. It appears to be a female Filaria. The worm measures 33 mm. in length by 2 mm. in width throughout the greater part of its length, but in life it must have been somewhat larger than this. The body of the worm is flattened from side to side and the colour is pale yellowish white. The cuticle is transversely striated, and throughout the whole length of the body longitudinal striae can be seen in the body wall. The anterior end is club-shaped and devoid of papillae and the mouth is situated terminally. At the posterior end there is a pair of rounded folds or prominences situated close together ventrally just in front of the extreme tip, which is somewhat truncated. In all probability the worm is an example of Filaria (?) *conjunctivae* Addario. There appears to be a considerable confusion in the literature regarding this worm and in consequence the classification must be regarded as doubtful. Similar, if not identical, worms have been from time to time recorded from a similar situation. Dubini was the first to record a case from the eye of a man in Italy, and Addario obtained a specimen from a cyst in the ocular conjunctiva. Another example was found by Alessandrini, in 1906. Braun gives *Filaria inermis* Grassi, *F. apabillocephala* Condorelli-Francaviglia and *F. peritonei hominis* Babes, as synonyms. In the case of this latter example the worm was found in the gastro-splenic omentum. Possibly the worm described under the name Agamofilaria *palpebralis* Pace in 1867, which was taken from a cyst in the eyelid of a patient is still a further example. Very little is known about this group; the term Agamofilaria, under which heading several different species are described, is not a generic name but "a group name for immature Filaria, the development of which does not admit of generic determination" (Fantham. Theobald).

SUB-CONJUNCTIVAL ADVANCEMENT

BY

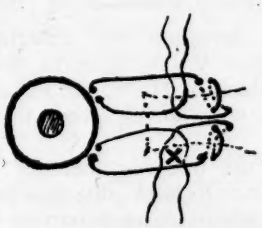
GEORGE YOUNG,

COLCHESTER

I have used the following simple process in advancing either the external, or the internal rectus muscle of the eye, in convergent or divergent strabismus, with or without muscular asthenopia, alone or in connection with tenotomy of the antagonist, with such good results, as to lead me to think it worthy of consideration.

A drop of pure adrenalin, 1 : 1000, is instilled, and repeated in a couple of minutes, whereupon the conjunctival sac is anaesthetized, with 2% holocain. Two instillations, about two minutes apart, suffice.

The speculum firmly fixed, a tiny opening is made in the conjunctiva and sub-conjunctival tissue, at x, with a straight canaliculus



knife, which has a convex sweep to its cutting edge, and which is passed through this opening and under the rectus tendon, the sclera is rasped from the tendon insertion for about ¼in. backwards, by turning the cutting edge towards it, and making half a dozen good to and fro movements with the cutting edge lightly pressed on to the sclera. Then the edge is reversed through 180°, and the under surface of the tendon and muscle belly are rasped in the same way for double the above distance, or more, as the case may require lesser or greater shortening.

Two strong silk sutures are inserted in double loops through the whole conjunctiva and muscle belly, one through its upper half, and the second through its lower half, and are again passed through corresponding points at the limbus, where not only conjunctiva, but half the thickness of the sclera is taken up on the point of the needle. The two sutures, tied and firmly drawn together, will tuck the muscle on itself. The forward half of its rasped under surface will be doubled on itself and will grow together, while the rest of its rasped under surface will adhere to that portion of the sclera which

has been also rasped. Both eyes are tied up for five days, and the stitches left in as long as one can. I leave them at least two weeks.

The sole disadvantage is the marked hump or pucker which is left by this tuck. I found it flattened down, however, and ceased to be at all unsightly in a few months. Everything else seems to me to be an advantage. Its simplicity, sub-conjunctival character, painlessness, speed, and above all its leaving the tendon uncut, and the chance of future greater advancement, if need be. Its effect is very powerful, and can be made more or less so by inserting the loops further back or further forward through the muscle, and by drawing the sutures more or less tightly together, on tying them.

The passing of the loops is a little painful, but even this can easily be avoided by an intramuscular injection.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

BEAUTIFUL weather favoured the International Congress of Ophthalmology held at Washington in the last week of April. The City of White Marbled Halls, statued monuments and shady avenues, decked out with spring green foliage, was looking its best.

Two days before the opening meeting the promoters of the Congress received a nasty shock, due to a fire breaking out in the New Willard Hotel, where its headquarters were situated, in what was to have been the meeting room. Some members of the Congress who had already taken up their residence at the hotel were awakened by telephone in the early hours of the morning, advised to dress quickly and descend by the elevators. The famous Peacock Alley of the hotel soon became crowded with guests in various degrees of negligée, holding hand luggage containing their most valued possessions. Fortunately the fire was confined to the tenth floor of the building, and was extinguished within an hour. Though the rooms in one of the other floors were rendered uninhabitable by damage from water, the members of the Congress who flowed into the hotel during the next few days were all accommodated.

The proposed meeting room in the hotel having been destroyed, the opening meeting of the Congress and the Scientific Sessions on the first three days were held in the spacious and beautiful hall belonging to the Daughters of the American Revolution. As this hall could not be utilized for lantern demonstrations during the day time, the final meeting was held at the building of the Medical Society of the District of Columbia.

At the opening of the Congress the call to order was made by Dr. Wm. H. Wilmer, the Chairman of the Committee on Arrangements.

An address of welcome was delivered by the Vice-President of the United States, Mr. Coolidge. The official delegates from different countries were then called and introduced. They were as follows:—

Argentine: Dr. F. J. Soriano.
Belgium: Dr. E. Gallemaerts.
Brazil: Dr. Cesario de Andrade and Dr. Pimental Franco.
China: Dr. T. M. Li.
Cuba: Dr. C. E. Finlay.
Habana: Dr. F. M. Fernandez and Dr. J. M. Penichet.
Czecho-Slovakia: Dr. W. Z. Hons.
France: Prof. F. de Lapersonne.
Great Britain: Mr. E. Treacher Collins.
Scotland: Dr. G. Mackay.
Italy: Dr. Salvatore Floria.
Netherlands: Prof. G. F. Rochat.
Peru: Dr. A. Beraun.
Sweden: Prof. A. Gullstrand.
United States: Lt.-Col. F. H. Garrison, *Army Medical Corps;* Lt. James F. Hooker, *Navy Medical Corps;* Surgeon J. McMullen, *Public Health Service.*
Venezuela: Dr. Santos Dominci.

Dr. Geo. E. de Schweinitz, of Philadelphia, was elected President of the Congress.

The afternoon of the first day was devoted to a most enjoyable excursion to the old house of Washington at Mount Vernon, where the President, on behalf of the Congress, laid a wreath on Washington's tomb. In the evening on the same day a reception of members and their ladies was held at the Corcoran Art Gallery.

Over 1,000 names were registered as members of the Congress, and though all the registered members did not appear, every meeting was very fully attended.

On the second day the members of the Congress were received by President Harding at the White House, with each of whom he shook hands. A most successful photograph of the assembled members, waiting to proceed into the White House, was taken by a rotating camera.

At the commencement of the final meeting of the Congress Mr.

E. Treacher Collins, on behalf of the Ophthalmological Societies of the British Isles, presented an invitation to hold the next International Ophthalmological Congress in London in 1925. Dr. Mackay, of Edinburgh, Dr. Rowan, of Glasgow, Dr. Gray Clegg, of Manchester, and Col. Elliot, of London, spoke supporting this proposition. Prof. E. Gallemaerts had come furnished with a similar invitation from the Société belge d'Ophtalmologie, but agreed to withdraw it in favour of that from Great Britain.

Prof. Gullstrand proposed the acceptance of Great Britain's invitation with the expression of a wish that at the next International Congress German should be recognized as one of the official languages; this was agreed to.

The proceedings of the Congress closed with a banquet held at the New Willard Hotel, over which Dr. de Schweinitz presided as toast master, with a grace and eloquence which it would be hard to excel.

All the toasts were responded to with enthusiasm, notwithstanding that the only fluid with which they could be pledged was iced water.

The whole proceedings of the Congress, both those of a scientific and those of a social character, were carried through in a way which manifested the most careful forethought and preparation.

The scientific proceedings of the International Congress of Ophthalmology consisted of 40 papers presented by members, four evening addresses, demonstrations of apparatus and methods of examination, and the exhibition of pathological specimens and microscope slides.

The forty papers were published in full in a pre-sessional volume, and short extracts of them in the three official languages were also issued in the form of a programme.

These papers included:—" The teaching of the war in ocular surgery," by Prof. de Lapersonne; " Detachment of the vitreous," by Sir William Lister, which was read for him in his absence, and which excited much discussion; " The diaphragm lamps in ophthalmology," by Prof. Gullstrand, the utility of which met with a chorus of approval from those who had employed them; " The ocular symptoms of poisoning by the sting of a scorpion," by Dr. J. de J. Gonzalez. Contributions:—" On the letters and characters of visual tests," by Dr. Ewing; "On anatomic *versus* ophthalmologic nomenclature," by Dr. M. U. Troncoso; and " On the standards for field taking," by Dr. L. C. Peter, led to the appointment of committees for report.

" The treatment of trachoma by sub-conjunctival injections of mercury cyanid " was discussed by Dr. Gemblath, and " Hereditary ocular degeneration " or " Ophthalmic abiotrophies," by Mr. E. Treacher Collins.

Prof. C. E. Finlay, and also a lady speaker, presented charts showing bitemporal contraction of fields of vision in connection with pregnancy.

A contribution by Dr. Moran, published in the pre-sessional volume on "The exchange of water and of ions through the cornea," and one by Mr. C. F. Harford on "Psychopathology in ophthalmic practice," were not read on account of the authors' inability to be present.

The operative treatment of strabismus was dealt with by Dr. B. Castrasana, and that of pterygium by Dr. E. Campodonico and Dr. S. L. Ziegler.

Dr. F. Poyales' paper on "Infantile ocular tuberculosis" was not well named as it dealt with two cases aged 9 years and one of 13 years. It excited, however, an interesting debate on intra-ocular tuberculosis in childhood.

The harmful effects sometimes met with when lens matter is left in the anterior chamber was dealt with by Dr. F. H. Verhoeff and Dr. A. W. Lemoine in a paper entitled "Endophthalmitis phaconaphylactica." Dr. A. E. Davis read a paper on "Serum and vaccine treatment for the prevention and cure of cataract." Prof. Barraquer described his method of removal of the lens in its capsule which he terms "Phacoerisis," and Prof. Gallemaerts presented an analysis of 46 operations performed in this way.

Dr. D. P. Kostitch described the ocular troubles which he had met with in 20 patients from gazing at the solar eclipse in Belgrade on April 9, 1921. Dr. A. Magitot advocated the superiority of the insertion of organic grafts of dead tissue, over that of unorganized tissue, to establish a mobile stump after enucleation of the eyeball.

Dr. A. F. Alonso and Dr. R. C. Cheney read papers on the treatment of ulcers of the cornea, and Dr. C. Charlin and Col. R. H. Elliot on glaucoma, the former dealing with its vascular conditions, and the latter with its diagnosis. Dr. Magitot and Dr. Bailliart recorded their work on endeavouring to find out the pressure of blood in the choroid vessels by means of a dynamometer, graduated in grammes, which is first applied so as to produce pulsation of the retinal vessels on the optic disc, and then with stronger pressure the disappearance of such pulsation.

Dr. J. M. Wheeler, in a paper on "Plastic operations about the eye," described the outcome of his extensive experience in this class of ophthalmic surgery, and Dr. J. N. Roy described an operation of blepharoplasty which he had performed in a case of bilateral lagophthalmos.

Papers on the technique of testing light and form sense were contributed by Mr. A. S. Percival, Dr. C. E. Ferree, Dr. G. Rand and Dr. E. Junés.

Dr. J. Grey Clegg read the description of some unusual con-

genital anomalies of the eyes. The effects of treatment with salvarsan in cases of interstitial keratitis was dealt with in papers by Dr. A. T. Estrada and Dr. A. Renshaw, and of treatment by vaccine of infectious keratitis by Dr. R. Virgilli.

Metastatic carcinoma of the choroid was described by Dr. A. Greenwood, and metastatic thyroid tumour of the orbit by Dr. Arnold Knapp.

A case of bilateral lymphosarcoma of the orbit with intermittent exophthalmos was described by Dr. W. S. Franklin and Dr. F. C. Cordes, and four cases of chloroma were summarized by Dr. A. J. Bedell.

The evening addresses were as follows:—

"Some descriptive errors in the anatomy of the orbit," by S. E. Whitnall, Prof. of Anatomy, McGill University.

"The clinical anatomy of the naso-lacrimal passage ways," by J. Parsons Schaeffer, Prof. of Anatomy Jefferson Medical College.

"The theories and realities of colour vision," by Leonard T. Troland, Prof. of Psychology, Harvard University.

"The production and hereditary transmission of certain eye defects," by W. G. Guyer, Prof. of Zoology, University of Wisconsin.

ANNOTATION

The Use of Light in Hospitals.

A most interesting paper was read by Mr. John Darch at the discussion upon the use of light in hospitals, arranged by the Illuminating Engineering Society.

Speaking first of the hospital ward, Mr. Darch advocated a light that should be quiet and pleasing, best obtained by a system of general lighting combined with local lighting. The general lighting need not be great, anything from one-half to one foot-candle, well diffused and without glare. The light should be spread evenly over the ceilings and friezes. Each patient should be provided with his own local light, giving him three foot-candles upon his book. This may be set on a short, smooth bracket close to the wall so as not to be in view of the patient. It should not be in the centre of the bed-head as usual, but about fifteen inches to the patient's left so as to avoid heat on his head and gloss on his book.

Local lighting is also necessary on the sisters' and nurses' tables, and each should have one or more well-shaded table-lamps adjustable so as to give an average of four foot-candles. The

decoration of the ward is an important factor in its illumination. Although ward and ceilings and walls are frequently to be found varnished, there should be no gloss above the dado. The ceilings and friezes should be white, the walls below are better of a quiet and restful colour, darker or lighter according to window space and aspect. As regards the operating theatre whatever arrangements are made the highest possible degree of asepsis should be maintained, yet the fittings suspended over the tables are often thickly coated with dust. One should admit the greatest possible angular expanse of glass without admitting direct sunlight, and the glass should extend nearly the length of the room. The ideal light for operations should be made to approximate to that found quite away in the open under a clouded sky.

The illumination should be not less than five-and-twenty foot-candles, and the light should be so thoroughly diffused that it should be difficult to get the shadow of one's hand upon the work. No exposed light sources should exist within the field of vision. The colour of the light should be as white as possible, and it must be uniform and steady. In special circumstances the surgeon can use an electric forehead light.

The author has seen nothing better for the purpose of illuminating the operating room than the white flame arcs we had before the war. He specially mentions the method more in favour abroad than here, viz., that of projected beams of light converging on the table from several points.

Mr. Darch closes his paper with a plea for the good lighting of dispensaries.

ABSTRACTS

I.—LIVING SUTURES IN THE TREATMENT OF PTOSIS

Wright, W. W. (Toronto, Canada).—The use of living sutures in the treatment of ptosis. *Arch. of Ophthal.*, March, 1922.

Stimulated by the studies of Dr. W. E. Gallie and A. B. de Mesurier on the use of living sutures in surgery, it appeared to **Wright** that living sutures would be an ideal method of treating ptosis. He operated by this method on eight eyelids in five patients. The fascia lata was exposed and cleansed by a longitudinal incision, five inches in length, made over the outer side of the thigh. Two parallel incisions were then made in the fascia lata, four to five mm. apart. The piece of fascia between these incisions was then freed from the underlying muscle. Before cutting this strip at its extremities it was slit longitudinally into the two required sutures.

The sutures were threaded at both ends into fairly large slightly curved needles with large eyes, and as the sutures were short and slippery it was necessary to tie them on to the needle with fine strong silk. Pagenstecher's operation was then performed upon the drooping lid. The operation was performed under the strictest possible aseptic technique. In spite of this four cases of Wright's series showed more or less infection, all of which, however, did well and in no case was the ultimate result thereby impaired. The author claims that in his operation we have a method of treating ptosis that is at least as good as any method previously introduced.

S. S.

II.—CHANGES IN THE CORNEAL ENDOTHELIUM FOLLOWING PARACENTESIS

Mazzei, Dr. A. (Naples).—On changes in the corneal endothelium following paracentesis of the anterior chamber. (Sulle alterazione dell'endotelio corneale in seguito alla paracentesi della camera anteriore.) *Arch. di Ottal.*, March-June, 1921.

Mazzei begins his paper by a review of previous observations on the changes that take place in the ciliary epithelium after paracentesis. As he was unable to find any account of the condition of the corneal endothelium in the literature of the subject he made a fresh series of experiments on rabbits. One eye of each animal was left untouched, and examined with the same fixation methods as the eye experimented on, to act as a control. The actual operation was performed as in an ordinary clinical paracentesis, and the author notes that the various eyes took very different times to empty. The eyes were excised at intervals of five, ten, and fifteen minutes, one, three, five, seventeen, twenty-four, and forty-eight hours after the paracentesis. The microscopical appearances are carefully described and illustrated by two drawings. His conclusions are as follows:—

1. Following paracentesis of the anterior chamber performed under the same conditions as in clinical treatment, certain changes, characterized mainly by vacuolization of the protoplasm, are found in the endothelium of Descemet's membrane.

The alterations in these cells are visible as early as five minutes after the operation, persist for several hours, and begin to disappear about seventeen hours; finally at the end of forty-eight hours the endothelium has recovered its normal aspect.

2. The alterations met with in the endothelium of Descemet's membrane closely correspond to those already observed in the cells of the pars ciliaris retinae.

E. E. H.

III.—STRABISMUS

(1) **Bettremieux.—The orthopaedic treatment of strabismus.** *La Clin. Ophtal.*, February, 1921.

Bettremieux has come to the conclusion that if squint in young children cannot be cured by glasses and non-operative measures it is better to operate, though he admits that the putting of the eye into the correct position does not guarantee binocular function. The operative method he employs is tenotomy of the rectus of the non-squinting eye. He avers that bad results have been due to bad operative measures, such as tenotomy of the squinting eye or double tenotomy at one sitting. He does not believe that advancements of external recti do less harm to the convergence apparatus, are more physiological, or are any less risky as regards overcorrection than tenotomy of the internal rectus of the fixing eye. On the other hand, he has never seen divergence or insufficiency of convergence resulting from tenotomy of the fixing eye, whereas the harmlessness of advancement from the overcorrection point of view is not as absolute as its partizans make out.

ERNEST THOMSON.

(2) **Schwartz, Frederick Oscar, M.D. (St. Louis, Mo).—Tenotomy and looping for the surgical correction of strabismus.** *Amer. Jl. of Ophthal.*, November, 1921, Series iii, Vol. IV, No. 11.

Schwartz gives a detailed description of an operation which he has found effective in dealing with both internal and external squint of considerable degree.

His method depends upon tenotomy of the opposing muscle, combined with advancement of the muscle which has to be shortened.

His aim is to anchor the latter muscle as firmly as possible, and he secures this by adopting the use of two thin gold plates, through which the sutures binding the muscle are threaded and tied, in order to act as a clamp.

Previous to operation precise measurements of the existing amount of deviation are recorded.

The opposing muscle is first divided completely so as to reduce the pull. The lips of the wound in Tenon's capsule and the conjunctiva are then drawn together in one suture.

Next, two double armed sutures, about 12 inches long, are threaded into a plate 7 mm. by 2 mm. rolled out of 14 c. gold wire, 4 small holes $\frac{3}{4}$ mm. apart being drilled in the plates.

Two of these plates are required for the operation.

One suture is threaded through the upper two holes of each plate, and the other suture in the lower holes.

The incision having been made in conjunctiva and Tenon's

capsule over the point of insertion of the rectus muscle, the muscle is raised upon a tenotomy hook and its attachment to adjacent tissue thoroughly freed by means of a second hook passed in various directions. The under surface is roughened by means of a scalpel. The muscle supported by the two hooks is now severed in a longitudinal direction for a short distance. Two silk threads are passed into the slit in the muscle and are used as retractors by an assistant, the gap in the substance of the muscle taking up a quadrilateral form. The amount of pull to be exerted by the assistant is determined by the degree of strabismus, a greater pull being demanded when the strabismus is marked and shortening of the muscle has to be more extreme.

The needles of the sutures already threaded through the gold plates having been introduced through the lips of the conjunctival incision, are passed in turn backwards through the adjacent band of divided muscle, and are then carried across the sharp angle formed by the retraction thread, to be re-introduced (from behind forwards) into the muscle near the point of insertion of the tendon. The needles are next passed out through this side of the conjunctival opening and threaded through the second gold plate in a similar fashion.

The sutures are now drawn together and the gold plates are in close proximity to one another on the surface of the conjunctiva. The sutures are tied, and the threads which were used as retractors are withdrawn. The gold plates act as a clamp and no further suture of the conjunctiva is required.

The roughened scleral surface of the muscle favours union of the tissue, and satisfactory anchorage of the muscle in its new situation should take place. The eyes are bandaged for eight to ten days, and the gold plates are removed in twelve days.

Clearly drawn diagrams assist in the description of this operation, which, provided one meets with a muscle capable of standing the amount of manipulation required, should give favourable results.

J. HAMILTON MCILROY.

IV.—KERATITIS CAUSED BY EXCESSIVE SUGAR INGESTION

Macleish, A. C., A.B., M.D. (Los Angeles, California). — Keratitis caused by excessive sugar ingestion. *Amer. Jl. of Ophthal.*, Vol. IV, No. 10. October, 1921.

Those who advocate the theory of the ill-effects of sugar on metabolism in cases of phlyctenular disease will take interest in the history of the case of keratitis reported by **Macleish.**

The patient was a girl, aged three years, white, who had suffered

from severe eye disease for one year. Constant treatment by different oculists had failed to produce improvement. The child was nervous, and her general health not good, constipation and diarrhoea alternating; and the appetite was poor and erratic.

Intense photophobia and lacrimation were present. There was some interstitial keratitis, but examination of the corneal condition was very difficult. There was no ulceration or phlyctenular conjunctivitis as far as could be made out. The Wassermann reaction was negative. On enquiry it was found that the child was allowed to eat an excessive amount of sugar. A strict diet, from which sugar was eliminated, was prescribed, and local treatment consisting of hot compresses and atropin. Two weeks later the child was seen and showed an almost incredible improvement. There was no photophobia or lacrimation; the corneae were clearer, and the pupils well dilated. The general health showed a corresponding change for the better. A month later the improvement was still more marked, and progress continued for several months. A relapse occurred during a temporary illness of the child's mother, when supervision of the diet was relaxed. The child, however, became well when attention was again directed to the diet. The condition appears to have been directly traceable to error in diet.

J. HAMILTON MCILROY.

BOOK NOTICES

Diseases of the Eye: a handbook of ophthalmic practice for students and practitioners. By GEORGE E. DE SCHWEINITZ. W. B. Saunders. 1921. Price, 50s. net.

This is the ninth edition of a deservedly popular handbook of ophthalmology. It has been entirely reset and revised, forms a handsome volume, and may be well recommended to the student and practitioner as a reliable and useful guide in his work.

Whilst conforming to the usual form of such text-books there are a few features that are outstanding and make the book more valuable.

The optics sections are very full and well treated, the explanations of the various problems are in simple language, and now that more knowledge is required from the student on this important subject should be of great value to those who are preparing for examinations. We would, however, criticise a good many of the diagrams, which are far from clear, and the lettering in most is obscure and small. There also appears to be some confusion in the definition of " range " and " amplitude " of accommodation, which seem to be treated as synonymous terms. Again, later in

the volume in the chapter on normal and abnormal refraction, the definitions of the various forms of ametropia contain the statement that there is disproportion between the " length " of the visual axis of the eye and the focal length of its dioptric apparatus. This is scarcely accurate.

The great value of the book is in the wealth of the clinical information which reflects the wide knowledge and reading of the author. In the chapter dealing with diseases of the uveal tract the relation of infections of the mucous membranes and absorption from septic areas is fully dealt with, thus bringing this chapter into line with present views.

The chapter on glaucoma is particularly clear and the mass of literature that has accumulated during recent years is well epitomised.

Diseases of the retina receive very full consideration, diseases of the blood vessels being well and clearly described. We find a good many of the illustrations in this section unconvincing and not very helpful.

Another excellent chapter is that which deals with operative ophthalmology, in which there is a full description of the various operations which have been recently introduced in the treatment of cataract and glaucoma.

In an appendix there are full details for the use of the ophthalmometer, tropometer and Dr. Sweet's apparatus for localizing foreign bodies in the eye and orbit.

There is a full and well arranged index.

Revista Cubana de Oftalmologia. Vol. III, Parts 1 and 4, Habana, Cuba. 1921.

Only the first and last of the four parts of this journal for the past year have reached us; the whole forms a bulky volume of nearly 900 pages. The general arrangement of the work is familiar to us, and that part of the volume in hand seems to compare very favourably with its predecessors; ophthalmology is evidently making giant strides in Cuba, and the punctual appearance of these large fasciculi reflects great credit on the editor, Dr. F. M. Fernández and on the Secretary, Dr. Jésus M. Penichet.

There is an interesting paper with a coloured plate by Edward Jackson on thrombosis of the central retinal vein following influenza. Barraquer describes his operation of phacoerisis; his paper is illustrated with the pictures which have already appeared in this country, and which will be familiar to all our readers. Santos Fernández contributes several short cases of clinical interest. Amat writes on atypical renal retinitis, and also contributes a valuable paper on chancres of the lids and conjunctiva. F. M. Fernández has a paper with coloured plate on a case of

multiple miliary aneurysms of the retinal artery in a man of 54 years of age, as well as a paper on conjunctivitis due to ipecacuanha and some shorter articles. The first fasciculus ends with a lengthy summary of statistics of cases operated on in the eye clinic at Santa Lucia, 1916-1920, and the customary *précis* of foreign and European ophthalmic literature.

We did not find the last fasciculus to be so interesting. Much of it is devoted to errors of refraction and to ionization in eye work, though there is a thoughtful paper by de la Garza on the prophylaxis of ophthalmia neonatorum, which is, of course, a very important problem for the welfare of the race.

A good deal of space is devoted to a long and able paper by Professor Finlay on the relations between ophthalmology and general medicine, illustrated by clear diagrams of the pons, medulla, floor of the fourth ventricle, and the anatomy of the cranial nerves.

NOTES

Honour. WE congratulate Mr. R. R. CRUISE who has received the honour of K.C.V.O.

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Elections. MR. A. L. WHITEHEAD (Leeds) has been elected President of the Section of Ophthalmology of the Royal Society of Medicine for 1922-23.

DR. A. MAITLAND RAMSAY has been elected President of the Royal Medico-Chirurgical Society of Glasgow for the Session 1922-23.

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British Medical Association AS readers are aware the Ninety-eighth Annual Meeting of the British Medical Association will be held at Glasgow, July 25-28. So far as the Section of Ophthalmology is concerned, the President will be Mr. A. S. Percival; the Vice-Presidents, Dr. A. J. Ballantyne, Sir William T. Lister, and Dr. John Rowan; the Hon. Secretaries, Mr. Percival J. Hay (Sheffield) and Dr. W. H. Manson (Glasgow). A discussion upon the "Aetiology of Optic Atrophy" will be opened by Dr. C. O. Hawthorne, Mr. J. Hogarth Pringle and Dr. H. Moss Traquair. On July 27 there will be a discussion upon the "Clinical Significance and Treatment of Heterophoria" to be opened by the President and Dr. L. Webster Fox (Philadelphia).

Oxford Ophthalmological Congress

SINCE the preliminary notice of the proceedings of the coming Oxford Ophthalmological Congress, given in our April number, the following additions have been made to the programme:—A discussion on "Methods of Operating for Cataract," to be opened by Sir Anderson Critchett. Included in the discussion will be a paper by Dr. K. K. Lundsgaard, of Copenhagen, entitled "Subconjunctival Extraction of Cataract," and also one by Mr. T. Harrison Butler on "Extraction of Cataract with Conjunctival Bridge Incision."

Other papers are by G. Russ-Wood "A Note on the Comparative Value of Artificial Illuminants," and by A. S. Percival (1) "A Note on the Light Sense," (2) "A Neglected Factor in the Aetiology of Miners' Nystagmus." Among the members coming from abroad we note the names of Dr. Lundsgaard, Dr. C. F. Bentzen, of Copenhagen, Professor Fritz Ask, of Lund, and Dr. O. H. Saunte, of Odensee, Denmark.

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Central London Ophthalmic Hospital Dinner

THE Annual Dinner of the Past and Present Students of the Central London Ophthalmic Hospital was held on June 8, 1922, at the Trocadero. Mr. T. Brittain Archer was in the chair, and between 30 and 40 were present.

The Chairman, after the toast of the the King, proposed the Hospital and its Medical School. He spoke of the improvement in the teaching of the school since its foundation fifteen years ago. Mr. Cunningham replied and said that the post-graduate students were not quite so many as before the War.

The health of visitors was proposed by Mr. Ernest Clarke. Amongst the visitors present were Sir John Herbert Parsons, Mr. J. Herbert Fisher and Mr. G. W. Thompson, and the toast was replied to by Mr. E. Treacher Collins.

The health of the Chairman was proposed by Mr. Mayou. He said that it was doubtful if the Central London Ophthalmic Hospital would be now in existence, if it had not not been for Mr. Archer. He had been connected with the Hospital since the year 1873, and senior surgeon for over 30 years and Chairman of the Committee of Management for nine years.

Mr. Levy proposed a vote of thanks to Mr. Neame and Mr. Druce for the successful organisation of the Dinner.

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F.R.C.S. with Ophthalmology

AT the first examination which has been held Dr. F. A. Williamson Noble has just achieved the distinction of being the first and only candidate to obtain the F.R.C.S. with Ophthalmology.